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THE EXPERIMENTAL PRODUCTION OF AMŒBIC DYSENTERY BY DIRECT INOCULATION INTO THE CÆCUM.

By Andrew Watson Sellards
and
Walter Albert Baetjer.

(From the Medical Clinic of the Johns Hopkins Hospital.)

OUTLINE.

I. Synopsis of the Usual Methods of Injection and their Results:
  1. Frequent transmission of amœbic dysentery to lower animals.
  2. Uncertainty of the result of rectal inoculation or ingestion of infective material in susceptible animals:
    A. 65 per cent of infections reported by Craig.
    B. 100 per cent failures reported by Walker.
  3. Distinction between first inoculation from man to lower animals and the subpassages from one kitten to another.
  4. Failure to secure continuous subpassage of a strain by ordinary methods of inoculation.

II. Methods of Inoculation.
  1. Explanation of failures by circumstantial conditions rather than individual non-susceptibility.
  2. Suggestion that pathogenic entamœbæ are unable to penetrate the uninjured mucosa.
  3. Failure of injections into wall of intestine.
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III. Results of Intracæcal Inoculations.
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  2. Comparatively high mortality from bacterial infections developing, usually, after several passages.

IV. Relation of Intracæcal Inoculations to Other Methods of Injection.
  1. Occasional failure of typical strains to infect, even at height of virulence, on inoculation per rectum.
  2. Failure of atypical strains of amœbæ to parasitize in animals, after rectal inoculation.
  3. Factors bearing on explanation of higher rate of infection on intracæcal inoculation:
    A. Anæsthesia.
    B. Lack of tendency to expulsion of injected material.
    C. Economy of infective material.
    D. Avoidance of deleterious diluting agents.
    E. Precision of the site of injection.

V. Relation of Bacteria to Amœbæ.
  1. Possible views that:
    A. Bacteria are beneficial for the growth of amœbæ.
    B. That they are an indifferent factor.
    C. That they are detrimental.
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VI. Application of Intracæcal Methods of Inoculation.
  1. Propagation of strains for investigation.
  2. Diagnosis of difficult cases.
  3. Study of atypical amœbæ.
  4. Study of the constancy of morphological characteristics of amœbæ.

Synopsis of Methods of Inoculation and Results.

The infection of lower animals with amœbic dysentery has been accomplished so frequently and with such typical reproduction of the principal features occurring in man that simple transference of the disease to lower animals ceases to be of special interest. Upon examining the methods of inoculation, however, it is found that the result of the injection of infective material in any individual instance is extremely uncertain. Even with the most suitable animals and with optimum infective material, there is a fairly high percentage of failures. Craig,[1] working with young kittens and with dysenteric stools, rich in trophozoites and cysts, obtained the best results by feeding material containing cysts, two-thirds of a small series of animals contracting dysentery. The most unfavorable results are reported by Walker[2]; three species of animals, namely, pigs, kittens and monkeys, remained persistently normal after inoculation with stools rich in amœbæ. Some of the monkeys were fed repeatedly with the cysts of *E. histolytica*, while others were fed, or injected per rectum, with dysenteric stools, in one instance the gastric juice being neutralized with magnesium oxide before feeding; in all, six monkeys were used. Two cats and a young pig were fed with cysts of the pathogenic entamœbæ, while six kittens were inoculated per rectum with trophozoites. In no instance, however, did any of these animals contract dysentery or even become parasitized. Wenyon[3] reported only four failures in twelve inoculations of *E. histolytica;* two of these failures can be disregarded, however, since they occurred under very unfavorable conditions; the other two occurred in an adult cat inoculated first with material obtained directly from a patient, and later with a strain that had been passed through two kittens. With two strains of *E. coli*, and also with an unidentified strain, no infections occurred. Moreover, in the case of *E. histolytica* it is important to note that this high percentage of infections was secured by Wenyon upon subinoculation from kitten to kitten; that is, the data are based chiefly upon successive transfers in lower animals. Since there is evidence that the virulence of the entamœbæ increased upon passage from animal to animal, one must distinguish carefully between percentages based upon the infections obtained in subpassages from kitten to kitten and the number of infections occurring in the first inoculation in lower animals.

Moreover, the results upon subinoculation have been so unsatisfactory that strains of dysentery have not ordinarily been carried through more than three or four passages in animals. In a single instance Werner[4] succeeded in carrying a strain through five, and in a second instance, through six passages. This difficulty in propagating any individual strain of dysentery in animals has usually been ascribed to the loss of virulence of the entamœbæ. Definite evidence was obtained by us, however, that it was due, in part, to the uncertainty of the method of inoculation.

[1] Craig: Parasitic Amœbæ of Man. Phila., 1911.
[2] Walker: Philippine J. Sc. B., 1913, VIII, 286.
[3] Wenyon: J. Lond. School Trop. Med., 1912, II, 27.
[4] Werner: Beih. z. Arch. f. Schiffs. u. Trop. Hyg., 1908, XII, 425.

Inoculation by Laparotomy into the Cæcum.

In any experimental work with dysentery it would be extremely desirable to obtain a method by which infection would follow with reasonable certainty upon the inoculation of virulent organisms. Very few suggestions have been offered, however, in explanation of the high percentage of animals which escape infection upon inoculation with virulent amœbæ. In general, the explanation of these failures has been referred, not to individual non-susceptibility, but rather to circumstantial factors. It has been thought by some that even virulent *E. histolytica* may frequently be unable to penetrate the uninjured mucosa of the intestine just as the protozoa and many bacteria are unable to penetrate the uninjured skin. On this basis we thought that more constant results might be obtained if the entamœbæ were injected into, or just under, the mucosa of the bowel, depending upon the resistance of the animal to take care of any bacterial invasion. Two monkeys and four young adult cats were inoculated under general anæsthesia in this way into the wall of the cæcum, the injection being made into the deeper coats of the bowel wall. The monkeys recovered promptly from the injection, no symptoms of dysentery developed, and at an exploratory laparotomy four weeks later, no ulceration could be found in the large intestine. Of the four cats, two died promptly from septicæmia, while the other two were sacrificed after two weeks, but showed no evidence of infection. This routine was abandoned and instead of inoculating into or under the mucosa, the infective material consisting of trophozoites, but apparently free from cysts, was injected into the lumen of the cæcum and the mucosa opposite the needle point was scarified and rolled with slight pressure between the thumb and fingers. The first three animals inoculated in this way developed typical bloody mucous stools containing many amœbæ after an incubation period of about one week. At autopsy, however, it was found that, in two instances, the lesions were distinctly below the lowest portion of the bowel which could possibly have been traumatized at operation. Accordingly, in the subsequent inoculations the material was injected directly into the lumen of the bowel with as little injury as possible to the mucosa. The details of the technique were as follows:

A laparotomy was done under general anæsthesia (ether) though care was taken to avoid the extremely deep narcosis resulting in sphincter relaxation and evacuation of the intestinal contents. Adult cats were selected when it was desired to secure chronic lesions but young animals, half-grown or smaller, were chosen when it was especially desirable to secure an active infection. In the first transfers from man to kittens no difficulty was experienced with bacterial infection.

As a routine the syringe was filled with the material to be inoculated before it was connected with the needle in order to have a sterile needle for puncturing the bowel; a moderately large puncture wound could be made in the gut, the syringe needle withdrawn and the peritoneum closed without further precautions. When flakes of blood and mucus were injected it was not necessary to comminute them since comparatively large pieces could be forced by moderate pressure through a medium sized needle. A considerable amount of pressure could be obtained when one person held the syringe needle firmly in position and the other attended to the injection, using preferably an asbestos packed syringe. Firm par-

ticles in the injection mass sometimes gave trouble by plugging the needle. When the material was not too viscous these particles could often be avoided by allowing them to settle out in the syringe for a few minutes, taking care to point the syringe upward during the injection so that they would gravitate away from the needle. When the material to be injected was especially troublesome, a small trocar on the syringe could be used with safety, provided the puncture wound was closed with stitches through the serosa.

No trouble from bacterial infection was experienced at any time in the inoculation of human stools into kittens. However, on subpassage from kitten to kitten there was a remarkable increase in the virulence of the accompanying bacteria. At times it became impossible to avoid peritonitis since, in some instances in young kittens, the bacteria were able to penetrate the bowel and produce peritonitis after injection per rectum. Fortunately, as the virulence of the bacteria increased, the virulence of the amœbæ also increased, so that the percentage of infections upon injection per rectum was comparatively high and it was only in the rarer instances that the animals were lost during the incubation period of the amœbæ by bacterial infection following rectal injection.

The large intestine in the cat is almost a straight tube lying practically in the mid line, the cæcum being rather rudimentary. The injections were made as a routine into the cæcum since it was supposed that the entire large bowel was susceptible to infection as in man. However, it was subsequently found that the lesions of the bowel appeared uniformly in the lower part of the rectum. The infections were taking place with such regularity that the injections into the cæcum were continued in order to give encysted amœbæ more time for germination. It is possible, of course, that the site of injection was in part responsible for the location of the lesions in the lower part of the bowel. In two instances, however, in which the injection was made a few inches above the ileocæcal valve, the lesions appeared as usual in the rectum and not in the cæcum. While this is only suggestive, nevertheless, we are inclined to feel that the absence of anatomical lesions in the cæcum was due, not to the site of injection, but that it was due perhaps to a natural non-susceptibility of this part of the bowel. Although the cæcum appeared to be the optimum point for routine injections, yet under special conditions one might want to consider other locations, such as the lower portion of the rectum.

The regularity with which the lesions appeared in the rectum after inoculation through the wall of the cæcum offers conclusive evidence that *E. histolytica* is able to penetrate the uninjured mucosa, or more precisely, that it is able to produce whatever injury is necessary in the healthy mucosa in order to penetrate it. The prompt healing of the puncture wound, although it was often thoroughly inoculated with amœbæ by back pressure after withdrawing the needle, and the development of the amœbic ulcers several inches lower in the bowel, were very striking. Furthermore, the scarification and maceration of the injected amœbæ into the mucosa of the intestine did not produce infection at the site of this injury.

Results of Intracæcal Inoculations.

The inoculations into the lumen of the cæcum gave unexpectedly favorable results. A variety of strains of amœbæ was tested, ranging from characteristic acute cases to extremely chronic atypical forms. These cases came from areas which were widely separated geographically; namely, the United States, Central America, and the Philippines. With the exception of those animals which died from secondary causes during the first week after inoculation, there were no instances in which the injection of amœbæ failed to infect the animals. This is especially interesting since some of the patients did not show characteristic symptoms of dysentery and the amœbæ in the specimens of stool used for inoculation were often extremely scarce and atypical in morphology. Eight strains of amœbæ were tested. One of these was a very acute relapse, two were moderately acute cases occurring in Panama City, three were chronic cases of very moderate severity from the Southern States, whereas the remaining two were cases of such an atypical nature that their etiology was not clear. In the original inoculation from patients to animals, a total of ten cats was used. All contracted dysentery with numerous entamœbæ in the stools. The incubation period varied from six to ten days, in acute and chronic cases, to one month in the animals inoculated with the atypical strains. We attempted to propagate three of these strains by subinoculation through a series of animals. In the subinoculations from kitten to kitten a total of nineteen animals was used for the intracæcal injections and ten of these died of bacterial infection, either peritonitis or septicæmia, within one to five days after injection; *i. e.*, during the incubation period of the amœbæ. The majority of these deaths occurred in a large group of animals which was inoculated with one strain, at a time when it was contaminated with an especially virulent streptococcus. Of the animals which lived six days or longer, all contracted dysentery. One strain was carried through five passages by the intracæcal inoculations and subsequently through six additional passages by inoculation per rectum; another through three passages; and a third was lost in the second passage by bacterial peritonitis. The positive results after increasing the virulence by passage are of less importance than the infections occurring in the first transfers from patients.

Relation of Intracæcal Inoculations to other Methods of Injection.

The general relationship of the intracæcal injections to other methods seems to be fairly clear. We made no attempt to secure any statistical data in regard to the comparative value of different methods. Two procedures for infecting animals have long been in common use; namely, the injection per rectum and feeding experiments. Neither of these procedures has consistently given more than 50% of infections, even under optimum conditions. Moreover, the results of inoculation have been so uncertain that it has not been possible to secure continuous propagation of amœbæ or to compare various strains of *E. histolytica* in regard to their pathogenesis or the fixity of their morphological characteristics. It is evident that it would be very desirable to have a method for studying the pathogenesis of unusual types of amœbæ, or even for diagnosis in suspected cases during the interim between

attacks. Although we have made no parallel experiments with exact controls, our experience with several strains of dysentery appears conclusive of the greater certainty of infection following intracæcal injections as compared with other methods. The following observations seem even more crucial than duplicate experiments under the same conditions. In the subinoculation of a typical strain of dysentery by rectal tube only two failures resulted in a total of eighteen animals.[5] These occurred, however, under conditions which illustrate well the uncertainty of injections per rectum. Plenty of material rich in trophozoites and cysts was used for these injections. The two failures occurred in kittens, one of which was three-fourths grown and the other one-eighth grown. Three other one-eighth-grown kittens of this same litter were successfully inoculated at the same time. Moreover, at the time that the second failure occurred, the strain had just been passed through a series of six kittens and its virulence had increased markedly, the incubation period being shortened from six to three days. That the virulence had not died out suddenly was shown by the infection of three other kittens at the same time, and the subsequent passage of this strain through a series of four animals without loss of virulence.

In contrast with these two failures, there was no instance in which the intracæcal injections failed to produce dysentery, even though the cases studied were extremely mild and the amœbæ in the specimens available for inoculation were extremely scanty and atypical in morphology.[6] The best illustration of this occurred in a patient with a history of diarrhœa of several years duration, without blood and with practically no mucus in the stool and without periods of intermission in symptoms. After purging with salts, microscopical examination showed three or four trophozoites and one four-nucleated cyst was found after looking over several coverslip preparations. The trophozoites were atypical in morphology and there were numerous uninucleated cells present which histologically might very well have been cysts of the limax group. Cultures for the limax group remained entirely negative. Inoculation of a rather large quantity of stool of this patient produced the same type of diarrhœa in a kitten, after an incubation period of one month, that characterized the symptoms in the patient. Subinoculations were readily secured. A second case was obtained with a similar clinical history. No motile amœbæ and no four-nucleated cysts were found microscopically, but the inoculation intracæcally of the stool of this patient produced a watery diarrhœa after an incubation period of one month, with the presence of moderate numbers of amœbæ, especially after purging.

The most favorable results with injections per rectum have been reported by Wenyon.[l.c.] Five strains of amœbæ were studied; two of these were typical pathogenic *E. histolytica*, one of which was inoculated successfully into two animals, the other failing to infect an adult cat. The other three strains produced the same type of diarrhœa in a kitten, after an incubation period of one month, that characterized the symptoms but all three of these non-pathogenic strains failed to parasitize cats. In subinoculations with the strain of *E. histolytica*, ten cats in all were used; only three negative results occurred and two of these were in animals inoculated with material from the fourth passage which was obtained several hours post mortem, most of the amœbæ being dead at the time of injection.

The result reported by other authors and our own experience have lead us to feel definitely that the intracæcal inoculation gives a better opportunity for infection than feeding experiments or rectal tube injections, and that it is distinctly preferable to some of the older procedures in which, after injection per rectum, the anus was sutured to avoid loss of the infective material.

The exact explanation of the favorable results after intracæcal inoculation probably depends upon a number of minor factors. At first glance it would seem that precisely the same result could be accomplished either by inoculation into the cæcum directly through its wall or by the use of a high rectal tube. Upon closer examination it will be seen that there are several minor points of difference. In the first place the general anæsthesia which is required is probably an advantage. It hardly seems possible that anæsthesia of a few minutes duration could have any appreciable effect on lowering the resistance of the animals. Its chief value in aiding infection probably lies in keeping the intestinal tract quiet and thus avoiding the prompt expulsion of the injected material. The animals after operation frequently remained as long as one or two days without defecating and then only formed stools were passed. Another point of definite significance is the economy of material which is permitted. It frequently happens that only a few flakes of blood and mucus rich in amœbæ and comparatively free from fæcal material, as in specimens obtained by the passage of a rectal tube, may be available. By an intracæcal injection this can be introduced directly into the lumen of the bowel with a minimum loss of material in the injection apparatus and often without the use of any diluting agents; at most only a minimum quantity of diluting fluid is necessary. This is a distinct advantage, for it is not possible to imitate artificially the exact composition of the menstruum in which the amœbæ are found, and they are rather better preserved in the medium in which they occur than in artificial mixtures of salt solutions. Lastly, the precision with which the site of injection can be selected is a factor to be considered.

[5] Certain minor precautions were observed throughout these inoculations. In the first place, the animals were always anæsthetized. This not only made them much easier to handle, but it prevented, in a large measure, the tendency to evacuation immediately after injection. A very small rectal tube was used, the smallest size catheter, cut to a convenient length and inserted rather high in the bowel. The infective material was injected with a syringe, using as little diluting fluid as feasible. After the injection, the catheter was compressed as it was withdrawn in order to distribute the material remaining in it throughout the bowel. A distinctly atypical case of dysentery inoculated per rectum with these precautions into a kitten produced a single acute attack.

[6] In one instance a negative result followed the inoculation of a specimen which showed no amœbæ after thorough microscopic examination.

With a rectal tube, one can only distinguish between a low, high and medium position. In view of the constancy with which the lesions appear in the lower rectum, it is quite possible that material containing only trophozoites could be inoculated to best advantage in this region. Moreover, with a rectal tube one cannot tell whether the blood and mucus is being introduced directly into a fæcal mass or against the wall of the intestine. With the intracæcal inoculation, the material can at least be deposited against the mucosa, avoiding an injection into definite fæcal masses.

Relation of the Amœbæ and Bacteria.

The question of the avoidance of fæcal masses during injection brings up for consideration the nature of the relationship of the amœbæ and bacteria to each other. All possible views have been suggested. It is almost an open question as to whether the bacteria are beneficial or detrimental to the growth of amœbæ, though possibly they constitute an indifferent factor. Perhaps the conditions vary with different species of bacteria, but it seems fairly certain that the bacteria seldom, if ever, constitute an indifferent factor. In many instances it was observed that the amœbæ were absent or very scarce in animals in which there was extensive bacterial infection such as peritonitis or septicæmia. Moreover, in the later stages of amœbic infection, the organisms frequently became much less numerous in those instances in which a profuse bacterial enteritis developed. Likewise, the bacteria were often extremely scanty in the profuse discharges of blood and mucus in the early stages of dysentery. Similarly, in an amœbic abscess of the liver, the bacteria, though present, were not sufficiently abundant to be identified by microscopical examination and could only be demonstrated by cultures. It would seem plausible to consider that bacteria, if present, must be in a large measure suppressed, since the unrestricted growth of bacteria is detrimental to the entamœbæ. It is more than probable that certain bacteria, in limited numbers, may serve as food for amœbæ; that bacteria are not essential is also shown by the occurrence of liver abscesses which are bacterially sterile. On this basis then, it would seem almost impossible for the pathogenic entamœbæ to grow in the normal fæcal contents of the bowel, but they could best secure their foot-hold on the surface of the mucosa. Of course, in active dysentery, when the fæcal matter is replaced by blood, mucus, and epithelial sloughs it would be quite conceivable that the amœbæ could multiply in this material in the lumen of the bowel as well as in the mucosa and submucosa.

Application of Intracæcal Inoculations.

It is evident that there is considerable opportunity for the use of more exact methods for the production of amœbic infection in animals. In our own experience, the use of intracæcal inoculation has afforded certain advantages over the usual methods. In the first place, we feel that it was in part responsible for the propagation of a strain of histolytica through a larger series of animals than has heretofore been obtained. It would seem to be a comparatively simple matter to keep actively infected animals on hand for at least several months, a period sufficiently long for studying many of the ordinary problems of dysentery.

In the usual routine of the examination of patients, the diagnosis of amœbic dysentery must rest altogether upon the clinical conditions and the morphology of the entamœbæ. Animal inoculation has not been of any assistance in the diagnosis of individual cases since the inoculation of pathogenic amœbæ too often fails to infect the animals. In two instances of obscure diarrhœa which were studied the symptoms of the patient were so unusual and the amœbæ were so scarce and so atypical that there was no justification for the diagnosis of amœbic infection as the cause of the diarrhœa. Animal inoculation showed definitely that the cases were due to amœbic infection. Here we have then the infection of animals with material so poor in amœbæ and so atypical in morphology that a clinical diagnosis is not possible. This is quite the reverse of the conditions in which an abundance of virulent organisms from acute cases fails to infect animals. It may even be suggested that inoculation into a susceptible animal may prove to be a more delicate method of diagnosis than the microscopic examination of the stools. This would be especially helpful when patients are seen in the interim between attacks or in the determination of the pathogenesis of cysts found in carriers of *E. histolytica*.

The exact position of these atypical strains requires somewhat elaborate study in order to classify them. These amœbæ differ much more both in the clinical symptoms which they produce and in their morphology than many protozoa which are accepted as distinct species. However, rather than describe a new variety, it seems better to designate them simply as atypical strains of *E. histolytica* until their exact position can be determined. With a more certain method for the infection of animals, it is possible to study these atypical forms more extensively, and to test experimentally, whether some of the morphological characteristics of the amœbæ which are supposed to be constant, may not really be subject to variation.

Lastly, it is self-evident that there are many features in the routine which we have followed in these inoculations which require modification. In the first place, it is desirable to determine exactly the optimum site of injection into the intestine, both for cysts and for trophozoites, and the exact value of injury to the mucosa at the site of injection. As regards bacterial infection, we feel that many of the difficulties which we have had could be avoided. However, extremely virulent strains of bacteria can make their appearance without warning; streptococci may appear at any time which are sufficiently virulent upon injection per rectum to cause a fatal septicæmia during the incubation period of the amœbæ. It is certainly desirable to devise some precise method of avoiding the complications of bacterial infection.

SUMMARY.

I. The inoculation of kittens per rectum, or the feeding of dysenteric stools rich in amœbæ has been used for the production of dysentery with widely varying degrees of success, but, in general, about 50% of infections are obtained. It has

not been possible by either of these methods to propagate a strain of pathogenic amœbæ beyond a very limited number of passages.

II. Inoculation of eight strains of dysentery, some of which were distinctly atypical cases, directly into the cæcum, in ten kittens produced an infection in all instances. In some of the subinoculations considerable difficulty was experienced with bacterial septicæmia.

III. Some of the factors bearing upon the high rate of amœbic infection following intracæcal inoculations are as follows:

1. Absence of tendency to expulsion of injected material.
2. Optimum utilization of infective material and the avoidance in a large measure, of deleterious diluting agents.
3. Precision of the site of injection in the bowel.

IV. Definite evidence was obtained that the amœbæ are able to injure and penetrate the healthy mucosa. Unrestricted growth of bacteria in the intestine seems to be unfavorable to the entamœbæ. A marked suppression of bacterial growth in the amœbic lesion appears to be necessary for the best development of the pathogenic amœbæ.

V. Animal inoculation by intracæcal injection has been of assistance in the

1. Propagation of a strain of amœbæ through a series of animals for a period of several months;
2. Determination of the etiology of some obscure diarrhœas;
3. Study of the morphology of some atypical amœbæ of low virulence.

ANALYSIS OF A CASE OF PSYCHASTHENIA.*

By Henri Flournoy, M. D.,
Interne, Henry Phipps Psychiatric Clinic.

The case which I wish to discuss is that of a man 28 years old, who suddenly developed the idea that he would have to kill his child. For five months this idea persisted without any change, then it disappeared and was replaced by the thought of killing himself. About two months after the development of this suicidal impulse the patient decided to come to the Henry Phipps Psychiatric Clinic for treatment.

A mental disorder of such a nature where one abnormal idea suddenly appears and remains in the mind of an apparently healthy individual is very similar to what occurs in cases of paranoia. The nature of such an idea, however, is different, as in the paranoiac it consists in a wrong interpretation of a limited part of the outside world—a systematic delusion not recognized as such. In the present case there was no delusion, but the patient referred to the ideas of killing his child or of killing himself as "harassed feelings" which he had to fight. He was conscious of his abnormal trend of thought, although he could not understand its mechanism. By analyzing his feelings and by trying to find out the why and wherefore of several incidents which had occurred before the onset of his obsession, when we found that he had once suspected his wife of infidelity and had sometimes doubted if the first child was really his, the idea of killing the child became much clearer in view of these facts which he had repressed.

In cases of this nature, it is advisable to take into consideration the slightest incidents preceding the onset of the disorder, and it is important to see clearly the inner conflict out of which the obsession might have sprung. By such a method of procedure our patients are put in the position of being able to understand the cause of their abnormal thoughts and to get rid of them. If they did not have such help, thoughts of an obsessive nature might cause more or less serious consequences, according to their nature and strength.

In 1846 Baillarger[1] reported the very striking case of a farmer, who, at the age of 17 years, developed the idea of killing his mother. He was unable to account to himself in any way for the onset of this idea. For 20 years he fought against it, and it was so strong that on two occasions he enlisted in the army so that he might be away from home. At the age of 37 this obsession gave way and was replaced by the idea of killing his sister-in-law. A few years later the patient consulted a physician, who gave him a certain amount of advice and bled him, but his frightful thought did not leave him. At the age of 43 he felt that he could not resist carrying out the act any longer, and to safeguard himself he begged his family and a physician to have himself locked up in an asylum.

From a diagnostic viewpoint we are dealing here with a type of obsession on account of which the patients feel, contrary to their conscious wish, a strong tendency to execute an act of the consequences of which they are afraid. Such a condition is closely akin to other obsessions characterized by doubts, impulses or phobias. Although disorders of this nature are usually described under the name of psychasthenia, yet they do not constitute a clean-cut disease, but should be considered as the results of previous and deeper difficulties. In each case an attempt should be made to understand the mechanism of the disorder by analyzing the concrete situations with which the patient had to cope.

S. H., a traveling man, 28 years old, was admitted to the Psychiatric Clinic in December, 1913. His father had died from chronic alcoholism, but otherwise the family history was negative.

Except for the ordinary diseases of childhood, the patient had always been in good health. He is stated to have been clever at school.

At home he was severely treated, especially by his father, who would not allow his children to have any recreation. He has, however, been a steady workman, having been employed by the same firm since his fourteenth year.

* Read at a meeting of the American Medico-Psychological Association, May 28, 1914.

[1] Annal. Med. Psych., 1846, 8, p. 10, quoted in Traite de pathol. ment., p. 752.

He married at 20. His wife and two children, both girls, 3 years and 8 weeks, respectively, are all healthy.

The physical condition of the patient is good. Apart from a slight irritability, there is no apparent mental trouble. His memory, his power of retention of recent impressions, and calculation are normal. There is no history of alcoholism. The patient is of a cheerful disposition, and has always been strictly devoted to the Roman Catholic Church, in which he was brought up.

His only complaint was that six months previous to admission he suddenly developed the fear that he might kill his first baby. During the month just preceding admission this fear faded away and was followed and replaced by the idea of killing himself. The patient consulted his physician and his confessor, and both of them advised him to fight against such ideas, but as he could not succeed, he got the impression that this thought of harming himself was a punishment for his sins.

On admission (Dec., 1913) the patient said: "I want to get rid of these worrying and harassing feelings, which I have had since June 2, 1913." On that date the patient had been in a southern city on a sales trip, while his wife and child had remained at home.

One morning on getting up feeling as well as usual the obsession, the onset of which he describes as follows, suddenly came to him: "After my bath, I stretched my arms out, a morning stretch, then *like a flash* the thought came over me that I was going to harm in some way or to kill my baby. This thought frightened me terribly and I tried to get rid of it, but I could not do it. Then I went out to work with this thought with me. I fought this way for five days, until I decided to come home, and I have been fighting it ever since."

In reviewing the history of his life, the patient gave the following important facts:

As a child, he had lived in constant fear of his father, who, being quick-tempered and sometimes drunk, used to beat his children and his wife. The patient had not only had a deep affection for his mother, but a feeling of pity and sensitiveness, as he frequently saw her maltreated or threatened. When he was 17 years old he left home to escape his father's supervision and to earn his own living. He soon began to satisfy all his desires, and indulged in sexual relations in an excessive manner until he married at the age of 20 years.

His wife had become pregnant by him four months before marriage, when they were engaged, and an abortion was performed in order to avoid a scandal. This episode created a deep religious conflict in the patient, as the abortion for which he was responsible was a sin strictly forbidden by the Catholic Church. Although he was forgiven by his confessor, his feeling of responsibility became unbearable. The patient gave an account of the conflict which ensued in the following words:

"At the time I always was convinced of my fault, but I would like to think that it was not my fault, on account of my responsibility to God. I never doubted that I was the cause of the abortion, but, *from a spiritual standpoint*, I would have been glad to hear that I had not been guilty, that means, that I could have excused myself possibly if I had been in the position to doubt my wife." So the patient was already willing, before they were married, to cultivate unjustified suspicions towards her, and the thought that another man might have been the cause of the pregnancy, and consequently of the abortion of his fiancee, was in keeping with his wish to avoid the responsibility of his act. He knew that his wife had never had anything to do with any other man, but he brooded over this suspicion to relieve his conscience.

After the marriage they lived at his mother's home. The patient then began to suffer greatly over the difference in the social standing of his mother and sister and of his wife, the latter belonging to a higher class. As he was very affectionate and touchy concerning his mother and sister, he felt the superiority of his wife's education with uneasiness and resentment. The patient stated very clearly that it would have given him some instinctive satisfaction if he had found that she was lower in some way. Here again the moral side was the only one where she might have been inferior, so that the patient's tendency to suspicion was increased after marriage, "always trying, he says, to find something wrong in her life, of which I could accuse and suspect her."

The only episode about which he thought he had ground to accuse her happened one and one-half years after marriage. One evening one of his friends came to his home to take him to the theater. As the patient was dressing his friend remained in the dining room in company with his wife. Suddenly the patient, who, from his own room, could scarcely hear their conversation, misunderstood a word and jumped at once to the conclusion that she had deceived him. "I just went up in the air. I was like a crazy man. I think the first thing I did was to tell this fellow that I couldn't go with him, I was sick. He left alone and then I started to fight with my wife. I remember that I accused her of everything under the sun, and that I pushed her violently down on the couch and accused her of having been untrue to me that afternoon with this fellow." His wife denied everything and all his further attempts to get any proof failed.

After he had worried one year about this matter without reaching any conclusion, he decided not to think any more about it and to banish it from his mind. Five years later, however, while in the hospital, he stated in a very frank manner that he had not allowed this idea of infidelity to come into his mind for several years. When advised to make this point clear, he answers: "I don't like to think of that," which is a good example of a voluntary repression, where the patient tried to keep away from his own mind the unclear and painful feeling of perhaps having been deceived.

It must be remembered that he himself, since marriage, in spite of his passionate nature and of the many temptations he had in his traveling profession, had always been true to his wife. In February, 1911, four years after the incident of suspicion, the first child was born, to the entire satisfaction of the patient and his wife. But when she was pregnant again in 1913 both were dissatisfied, because for financial reasons they did not want another child so soon. The question of an abortion arose again, but was declined at once, for the patient would not burden himself with this guilt a second time. Following this he became nervous and irritable, but accepted the idea of having a second child up until June 1, 1913, when a new incident brought back the old difficulty of the suspicion towards his wife, which for five years he had not thought about.

Towards the end of May, 1913, he had met a young girl on the trip already mentioned, whose company he enjoyed. On May 31, 1913, she told him that she had had relations with a friend of his, and following this he brooded and felt ashamed of his friend, who had been untrue to a faithful wife. He also experienced a feeling of jealousy, as he himself had had a strong liking for the girl, which, out of respect for his own wife and child, he had to master.

The next morning, June 2, the thought of killing the first child flashed into his mind. This obsession gradually disappeared, when at the end of October the second child was born, but only to be replaced by the fear that he would kill himself.

He describes the obsessive character of such fears, when he says: "'The impression would come to me that I could not do such a thing, then instantly the thought would follow that no matter what I did to fight against these feelings, the inevitable result would be, that I would do it."

He fought several months, but never tried to find the reasons which could explain such thoughts. He did not think that they might have any connection with previous causes of worry, such as the suspicions he had had towards his wife; however, he cooperated in the review of the whole situation which revealed the above data.

At this point in the analysis the probable mechanism of the obsession was assumed to be as follows:

While ruminating over a possible intrigue with the girl the conflict between his instinctive impulse and his duty as husband and father was unusually strong, and the suspicion perhaps that he had once been deceived by his wife would have come into his mind, if he had not been in the habit of banishing it. He did not think of it. This old question was again repressed, but the situation was too hard to cope with, and the smaller and more recent difficulty of his married life, viz., the idea of soon having a second child, helped to determine the form of the obsession, that is, the frightful idea of killing his child, replaced later by the fear of killing himself.

The above explanation was given to the patient on the 4th of January, and on the 5th he felt relieved. The obsession to kill himself did not come any more, and when he thought of it there was no fear.

Six days later he again began to have a slight feeling of uneasiness concerning his first child, with fear of harming it. A deeper analysis of old memories and of his dreams showed still more definitely how this obsession was closely connected with his suspicions of his wife. The patient had doubted sometimes if this child was his, although he had never allowed this doubt to remain in his mind.

When compelled to reason out the situation, he finds that the only man whom he suspects his wife to have been with died at least one year before the child was born, and he says: "While not for one second I ever gave it a thought to question the birth of either one of my babies, I feel relieved to know that they are both mine, especially my first baby, because I have absolute proof."

In view of this revelation of the patient's latent doubts as to the paternity of the children, chiefly the first one, the probable mechanism of his obsession became somewhat clearer. His fear of harming the child disappeared again.

Discussing also why he had once accused his wife of having deceived him, he realized how blindly he had jumped to a conclusion; the more he thought of it the more he got convinced of her faithfulness, whereas the only suspicion could be the word which he misunderstood. "All these reasons tend almost to give me an absolute proof that I have been mistaken. I never tried before to reason this whole thing out in any way. When the matter came to my mind, at first, I asked my wife over and over again, and she denied it always in the same frank manner; however, I was never convinced because I never reasoned it out myself. Later I banished this question when it came. I have not had an occasion to suspect her afterwards."

This statement shows what a strong tendency the patient had to suspect his wife. "Before meeting her, my impressions of women were that they were very weak, easily persuaded to do wrong, but I did not realize that men were just as weak. I had always been kept down at home, not allowed to have much company and thus got this impression of women from my father's treatment of my mother."

Such an explanation is only very superficial, but since it comes from the patient, it shows that he realizes how far back the roots for his suspicious state of mind are to be found in himself, more than in the behavior of his wife.

The patient left the Clinic February 14, 1914. He felt perfectly well until March 10, when a slight difficulty with his wife made him feel a little uneasy about his children. He got quickly over this trouble and felt well again.

The striking points in this case are:

(a) The sudden onset of an obsessive fear that he would kill his child, then that he would kill himself, against which the patient fought in vain for seven months.

(b) The disappearance of the obsession as soon as the patient saw that it could be connected with previous difficulties.

(c) The persistence of an uneasy feeling towards the children, without fear, arising at times when the patient is depressed or crossed.

The whole trouble is closely related to a suspicious state of mind, a fact which the patient had to realize. His prospect of avoiding a relapse of the obsessive fears depends greatly on the extent to which he will be able to assume a new mental attitude towards his wife.

FIBRINOLYSIS IN CHRONIC HEPATIC INSUFFICIENCY.*

By E. W. Goodpasture, M. D.,

Instructor in Pathology, Johns Hopkins University.

(From the Hunterian Laboratory of Experimental Medicine, Johns Hopkins Medical School.)

In the course of a study of the blood in hemorrhagic conditions a case of very advanced hepatic cirrhosis came under observation. The patient had been bleeding frequently and freely from the nose, mouth and rectum. Her condition was extreme and shortly she came to autopsy. Blood was drawn from the heart soon after death. It clotted firmly in normal time. Three and one-half hours later the clot had completely dissolved, leaving no trace of fibrin.

Following this we observed specimens of blood from many patients with various diseases to see if any clot would undergo a similar dissolution.† The study has included four cases of atrophic hepatic cirrhosis, which have come to autopsy; and it is the interesting phenomenon of "fibrinolysis" in their blood which constitutes the subject of this paper.

The term "fibrinolysis" was first used by Dastre,[1] in 1893. He coined the word to designate the phenomenon of fibrin dissolution in serum. Whipped fibrin left in its own serum 18 hours lost on an average 8% in weight. Dastre called attention to this as a possible source of error in quantitative estimations of this proteid. He worked with the blood of dogs.

Despite this initial loss in weight of fibrin a clot from normal blood will not undergo complete dissolution for a very long time. It may be incubated at body temperature for days, or weeks even, without entirely liquefying, if kept sterile. In fact it is a matter of days, as a rule, before one can detect with the eye any change in the clot. This extremely slow autolysis

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of blood has been long observed. Under certain experimentally induced conditions, however, fibrinolysis may become very active. For instance, Jacoby,[2] in 1900, studying the relation of changes in liver and blood in phosphorus poisoning to autolysis, observed that blood from a severely poisoned dog was actively fibrinolytic. Blood drawn from this dog at the height of intoxication would not clot, owing to an absence of fibrinogen; but when normal blood was added to it the mixture clotted readily. After a while the clot was completely dissolved. Other observers have called attention to active fibrinolysis in the blood of phosphorus poisoned animals, but rather incidentally as Jacoby did.

In 1905 Nolf[3] discovered fibrinolysis active in the blood of dogs which had received intravenous injections of proteoses. He observed the same condition likewise in animals after extirpation of the liver.

Years before Dastre called attention to fibrinolysis in serum, the fact that fibrin dissolves in dilute solutions of certain salts, like KNO_3, NaCl, $[NH_4]_2$ SO_4, Na_2SO_4, was well known, and had given rise to much work and discussion.[4] The outcome of these investigations was the proof that dissolution of fibrin under such conditions was not due to putrefaction, and the probability that it resulted from proteolytic activity of enmeshed leucocytes, rather than from a simple physical dissolution without chemical change—a view held by Hammarsten and others. Dastre himself believed the dissolution of fibrin due to a process of digestion, for he found proteoses and peptones were formed as a result of it. He imagined the salts acted like a proteolytic ferment. Rulot[4] upheld the leucocytic theory of digestion, and showed that when the white corpuscles were eliminated very little digestion of fibrin took place.

The problem of an active fibrinolysis in serum is even more complex and difficult to explain satisfactorily. For here one has to deal with another factor, namely, the well-known antiproteolytic property of blood. Granting the dissolution of fibrin in serum to be due to enzyme activity, two main possibilities suggest themselves to explain it, viz., (a) great increase of proteolytic ferment overbalancing the normal antagonistic property of the blood, and (b) a diminution or absence of antiproteolytic substance, permitting the normal proteolytic ferment to act.

Morawitz,[5] in 1906, studying post-mortem changes in human blood, noted the rather frequent occurrence of fibrinolysis of intravascular clots in bodies autopsied sometime after death. In one case the blood was completely fluid and no fibrinogen could be demonstrated as soon as ten hours after death. Morawitz states that fibrinogen can be destroyed, probably by fibrinolytic ferment, before any clotting occurs post mortem. Of his cases those that met a sudden death were more likely to exhibit post-mortem fibrinolysis.

Aside from the statement that blood in severe cases of leukemia may become proteolytic,[5] I have found no reference to the ante-mortem occurrence of pathological fibrinolysis * in human disease. Recently Saxl[7] investigated the proteolytic power of normal and pathological human sera toward peptone. After two days, the shortest interval of digestion in his experiments, there was some proteolysis by the serum of each of the 26 cases studied, including a case of cirrhosis hepatis. The difference in amount of digestion was not striking.

In the following cases of cirrhosis fibrinolysis has been very rapid and clean cut:

CASE I.—Med. Hist. No. 31530. B. H., age 55, white female. Admitted to J. H. H. October 1, 1913. Complaint: pain in stomach, shortness of breath. F. H.: negative. Headaches for several years, short of breath for two years. Has had bloody stools for two years. Marked alcoholic history, usually 12 to 18 bottles of beer a day, since the age of 16.

P. I. began three months ago with enlargement of the abdomen. Skin yellow for four months. Occasional epistaxis. Vomiting of greenish material with no loss of blood. Day before admission patient began to be mentally dull. P. E.: scleræ have yellowish tinge. Heart dullness 14 cm. to the left; soft, blowing systolic murmur. Abdomen distended, numerous large veins over the lateral portions. Small area of tympany in the umbilical region. White blood cells 21,800. Hb. 70 per cent. Day of admission tapping was done; 6½ liters of turbid, yellow fluid removed, containing 2200 white blood cells per cu. m. Sp. Gr. 1012. Palpation after tapping, liver edge just felt; dullness to the costal margin; no masses; tenderness.

October 3d. Patient has been comatose since admission. Definite impairment of percussion note over the right base up to the 6th spine of the back. Breath sounds feeble. Numerous rales. Tendency to bleed rather freely from nose, gums and rectum.

Impression: Cirrhosis of liver, toxic stage; bronchopneumonia of right lower lobe. Urine: Sp. Gr. 1012, trace of albumin. No casts.

Anatomical Diagnosis.—Cirrhosis of liver; chronic fibrous tuberculosis (apex of left lung); acute tuberculous bronchopneumonia (apex of left lung); tuberculous peritonitis; chronic diffuse nephritis; hemorrhagic and diphtheritic gastritis; chronic splenic tumor; bronchopneumonia; bronchitis; tracheitis; jaundice; bilateral ovarian cyst; bilateral hydrosalpinx; ulcerative vaginitis; tuberculous bronchial lymphadenitis (caseation); chronic fibrous pancreatitis; chronic tuberculous perihepatitis and perisplenitis; accessory spleen.

Body is that of an obese white woman, 155 cm. long. The pupils are equal and not dilated. The conjunctivæ, mucous membranes and skin, especially of the face, show a marked icteric tint. There is a puncture wound from tapping in the midline below the umbilicus. The abdomen is rather full and slight fluctuation is made out. Lineæ atrophicæ are present on the abdomen. The right leg is 8 cm. shorter than the left. This shortening is in the thigh. The femur is bent and thickened. The bend occurs about the junction of the middle of the lower third. The right lower leg has a number of old cutaneous scars. On incising the body there is abundant panniculus. The abdominal cavity contains a considerable excess of yellow, slightly turbid fluid. The omentum is thickened and laden with fat, and is slightly rolled up over the transverse colon. The blood vessels of the mesentery and peritoneum are injected, and over all the peritoneal surfaces are thickly scattered, small, translucent, slightly elevated granules about ½-1½ mm. in diameter. Some of these have a yellowish center and practically all of them have a slight halo of injection around them. These granules are distributed everywhere but are perhaps more numerous in the flanks and pelvis and also in the more

* Since the clot from any specimen of blood will in all probability undergo a certain amount of dissolution if left in its serum a sufficient length of time, it becomes necessary to define what one means by a pathological fibrinolysis. From our observations we have concluded that any blood whose clot dissolves completely in 12 hours at body temperature is pathological.

exposed portions of the mesentery and intestinal coils, although this distribution is not very marked. The liver is small, the edge lying slightly above the costal margin, and it is very firm. The gall bladder is adherent to the colon and omentum, and there are numerous adhesions about the pelvis, where a large, rounded, cyst-like body projects upward above the pelvic brim on the right side. On opening the chest it is found that the diaphragm has been pushed upward, to a slight extent lessening the size of the thoracic cavity. The heart lies in a normal position. The pleural surfaces are smooth and glistening.

Liver weighs 1425 gm., measures 23 x 14 x 8½ cm. It is of a very remarkable firmness, resembling hard rubber. The outer surface has a few small, opaque granules as described above. Aside from these it is very rough. The lobules are outlined by depressions. It is also mottled in color; the centers of the lobules appear to be yellow, while the remainder is of a darker tinge, while in some places there is grayish material resembling connective tissue. On section the lobules are readily seen. The liver tissue, however, seems to be compressed into a very small area. In every case it is bile stained. The portal zones are very thick and seem to be composed mostly of grayish, translucent looking tissue. Throughout them are many tiny blood vessels which in places give the portal zone a purplish appearance. In a few places are seen tiny, yellow, opaque dots, distributed without relation to the lobules.

Microscopically the liver shows well marked but not extreme annular cirrhosis. Central congestion and fatty change. Advanced repair. In connective tissue, well marked mononuclear infiltration and vascular congestion.

Blood Examination.—Blood was drawn from the heart one hour after death. It was hydræmic and had not yet begun to clot intravascularly. Five minutes after withdrawal it clotted firmly. A portion of blood was drawn into 1 per cent sodium oxalate to prevent clotting. This was centrifuged, and 20 cc. of the supernatant plasma used to determine the amount of fibrinogen. The plasma was bile stained and faintly alkaline to litmus. A part of this plasma was used for coagulation tests and tests for fibrinolysis.

The coagulation time was normal:

1 cc. plasma + 1 drop $CaCl_2$ (1%) = clot 6 minutes.

The original clot from the heart's blood and specimens of clotted oxalate plasma were placed in the thermostat at 37° C. In three and one-half hours they were fluid again. The clots had completely dissolved.

The plasma was capable of digesting dog's fibrin in small amounts, but was inhibited by dog's plasma in larger amounts.

1. .25 cc. human plasma + .75 cc. dog's plasma + Ca 2 drops = clot = no fibrinolysis 24 hours.
2. .5 cc. human plasma + .5 cc. dog's plasma + Ca 2 drops = clot = partial fibrinolysis 24 hours.
3. .75 cc. human plasma + .25 cc. dog's plasma + Ca 2 drops = clot = complete fibrinolysis 4 hours.

Inhibition of fibrinolysis by dog's serum.

1. .5 cc. human plasma + .5 cc. dog's serum + Ca 1 drop = clot = complete fibrinolysis 15 hours.
2. .5 cc. human plasma + .5 cc. normal salt solution + Ca 1 drop = clot = complete fibrinolysis 3 hours.

Tests made with oxalate plasma on three successive days showed a marked diminution in fibrinolytic activity. Whereas the clot digested in three and one-half hours on the first day, it digested in nine hours on the third day. After heating clotted oxalate plasma at 60° C. for 30 minutes no fibrinolysis took place within 12 hours.

Fibrinogen estimation yielded the low quantity of .183 gm. per 100 cc. blood plasma. This is about one-third the normal fibrinogen content of human plasma.

SUMMARY.—White woman, age 55, with ascites, leucocytes 21,880, jaundice and bleeding from nose, gums and rectum, came to autopsy. Liver showed typical atrophic cirrhosis. Blood drawn from heart immediately after death clotted in normal time. The clot dissolved completely in 3½ hours. Digestion of clot was inhibited by the addition of dog's serum to oxalate plasma. Heat at 60° C. for 30 minutes also inhibited digestion. Fibrinogen content of this plasma was .183 gm. per 100 cc.

CASE II.—Med. Hist. No. 31476. Autopsy No. 4024. G. T., age 47, white male. Admitted September 19. Complaint: swelling of abdomen and legs. Always in good health. Typhoid fever 15 years ago. Has had bleeding hemorrhoids for 15 years. Marked alcoholic history.

P. I. began four months ago with tenderness and swelling of abdomen, dyspnoea. Bleeding from hemorrhoids.

P. E.: On entrance icterus, collateral circulation noted. Impairment at both apices. Oedema of scrotum and legs.

Clinical Impression: Cirrhosis of liver with pulmonary tuberculosis. Blood count 10,000 white cells. Hemoglobin 62 per cent September 20. Coagulation time longer than normal. Examination of larynx (Dr. Crowe) showed reddening and granulation. Diagnosis: Tuberculosis probably. Wassermann negative.

October 3d. Abdominal tapping. October 5th. Rise in temperature to 104.8° and signs of pneumonia in right side. October 23d. Tetrachlorphthalein test gave 40 per cent output.

November 3d. Red blood cells, 1,952,000. White blood cells, 5600. Hb. 35 per cent.

Anatomical Diagnosis.—Diffuse cirrhosis of liver (atrophic type); chronic pancreatitis; passive congestion of abdominal viscera; chronic fibroid apical tuberculosis; chronic laryngeal tuberculosis; general anasarca; emaciation; bronchopneumonia (organizing); congestion and œdema of lungs; acute aortic endocarditis; acute splenic tumor; disseminated ecchymoses; intramuscular hemorrhage (puncture wound); bilateral inguinal hernia; blood abnormality—fibrinolytic ferment and low fibrinogen content; chronic gastritis; chronic ulcerative laryngitis (tuberculous).

Body is that of a large framed, somewhat emaciated white male, 179 cm. long. Rigor mortis is present. Icterus is evident over the neck and chest. The right arm and lower extremities show notable boggy œdema, and the subcutaneous tissues pit deeply on pressure. The scrotum is very œdematous and on incision is found to contain two large hernial sacs, the left being larger, its orifice admitting three fingers with ease. The short loop of the sigmoid is adherent by pretty tough adhesions to the neck of this left inguinal hernia. The testicles on section appear normal. On section through the abdominal wall one opens into a cavity containing bloody fluid. This is just below the puncture wounds (abdominal tapping). The extravasated blood had been completely digested, soft and fluid. It had burrowed laterally in the sheaths of the recti and in the properitoneal fascia, extending down in the pelvis in the neighborhood of the bladder and representing in all perhaps 100 cc. of blood. The abdominal cavity contains about ½ liter of clear, lemon yellow fluid. The peritoneal surfaces are smooth, but show milky patches of thickening, more especially about the root of the mesentery. The omentum and mesentery are rich in canary colored fat. There are a few adhesions about the spleen, but the liver is free. The thorax is barrel shaped. On opening the pleural cavities they contains an excess of clear, lemon yellow fluid. There are dense adhesions over both apices. The pericardial sac contains an excess of fluid, slightly blood tinged, due to the aspiration of blood shortly after death.

Liver weighs 1480 gm. It presents the typical picture of Lænnec cirrhosis. The capsule is thickened and rather milky. The surface is coarsely granular, showing warty areas sticking above the surface. Collateral circulation in the suspensory ligament is very conspicuous. The gall bladder is dilated, thin walled, but the bile passages are normal. On section parts of the organ show very

extensive scarring, whereas other portions show a more diffuse and not so extensive involvement. There are nodules and lumps of liver parenchyma of all sizes between the scars. These nodules may be gray or pigmented yellowish-brown. There are bright red areas apparently associated with hemorrhage and perhaps necrosis, in some of the liver cells. The architecture is greatly obliterated. The left lobe is thin, flattened and elongated, but in general presents the same changes found in the right lobe. The organ is very firm and hard. The mucosa of the gall bladder is normal.

Microscopic Sections.—Liver shows the usual picture of diffuse cirrhosis in which dense connective tissue is not very conspicuous. The mononuclear cell reaction is quite pronounced. Icterus is present in some lobules. A good deal of liver epithelium is well preserved and only slightly distorted. Some liver cells show hypertrophy. The new formed bile duct reaction is not striking. There are some focal necrosis. The necrotic cells are often bile stained.

Blood Examination.—Blood from this case was first examined October 21, 1913, one month before death. It was quite hydræmic. At this time the fibrinogen was .3435 gm. per 100 cc., just below the limit of normal. Blood drawn from an arm vein clotted firmly in normal time. Clots from whole blood and from oxalate plasma were placed in the thermostat at 37° C., and at the end of 16 hours were completely fluid. The specimens were not examined at intermediate intervals, consequently the exact time of complete dissolution is not known. In this case fibrinolysis was inhibited by cat's plasma in all dilutions used up to equal parts.

1. .5 cc. cat's plasma + .1 cc. human plasma + Ca 1 drop = clot = no fibrinolysis 24 hours.
2. .5 cc. cat's plasma + .5 cc. human plasma + Ca 1 drop = clot = no fibrinolysis 24 hours.

Oxalate plasma kept on ice 48 hours, then clotted with calcium and incubated, showed only slight dissolution after 24 hours. There was also a definite diminution in fibrinolytic activity in plasma two or three days old, as in the previous case. Phenoltetrachlorphthalein excretion in the feces at this time was normal, 30 per cent. Blood lipase was also normal.

One month after the above tests were made this patient came to autopsy. Blood was drawn from the heart within one hour after death. It was very watery, the cellular elements constituting only one-tenth or less of the total volume. The blood clotted five minutes after aspiration from the heart. The clot was jelly-like but firm enough to permit inversion of the flask without breaking. The fibrinogen had fallen to .0975 gm. per 100 cc.

Clots from whole blood and from oxalate plasma coagulated with calcium and with thrombin, dissolved completely within three and one-half hours at 37° C.

The following tests were made with fibrinolytic serum plus normal human plasma. The human plasma was obtained by receiving blood into iced paraffined tubes, centrifuging and pipetting off the supernatant fluid.

1. .25 cc. serum + .75 cc. normal plasma = clot = no fibrinolysis 24 hours.
2. .5 cc. serum + .5 cc. normal plasma = clot = no fibrinolysis 24 hours.
3. .75 cc. serum + .25 cc. normal plasma = clot = no fibrinolysis 24 hours.

These clots showed no fibrinolysis after incubating for three days, illustrating the inhibiting effect of normal blood.

No digestion of egg albumin was observed in Mett's tubes incubated in this fibrinolytic serum 10 hours at 37° C.

As in the previous cases fibrinolytic activity diminished greatly in plasma kept three or four days. The plasma used in the following experiment was three days old.

1 cc. plasma + Ca 2 drops = clot = partial dissolution 8 hours; complete 24 hours.

On the first day the clot dissolved in three and one-half hours.

SUMMARY.—White man, age 47, with ascites, jaundice, loss in weight, expectoration of blood, active bleeding piles, and leucocytes 5200, came to autopsy. The liver exhibited atrophic cirrhosis. One month before death blood drawn from an arm vein clotted in normal time. The clot dissolved completely in 16 hours, perhaps sooner. The fibrinogen content of the blood plasma was .3435 gm. per 100 cc. Serum lipase was normal. Phenoltetrachlorphthalein output in feces was normal, *i. e.*, 30%.

Blood drawn from the heart immediately after death clotted in normal time. The clot dissolved completely within 3½ hours at 37° C. Oxalate plasma clotted with normal thrombin dissolved its clot in the same time. Digestion of the clot was inhibited by normal human serum. Mett's tubes incubated 10 hours at 37° C. in fibrinolytic serum showed no sensible digestion. After three days clots formed by adding Ca to oxalate plasma, digested in 8 hours. Fibrinogen content of this plasma was .0975 gm. per 100 cc.

CASE III.—J. H., colored male, age 50. Admitted to Bay View October 8, 1913. P. I. began two weeks before admission with pain in left side, and swelling of both legs. Bleeding piles four years ago. P. E.: Ascites. Distention of abdominal veins. Occult blood in stools. W. B. C. 6000. Hb. 65 per cent. Died January 18, 1914.

Anatomical Diagnosis.—Atrophic cirrhosis of liver. The liver was lobulated in gross and under microscope showed diffuse increase in connective tissue, and hypertrophic nodules of liver cells.

Blood Examination.—On December 13, 1913, this patient was bled. On the following morning, 13 hours after its formation, the clot was completely dissolved. Tests were then made with oxalate plasma. Clots formed normally on the addition of calcium and completely dissolved when incubated four hours at 37° C. At the same time these tests were made specimens of clotted plasma were heated on a water bath at 60° C. for 30 minutes, then incubated. They showed no fibrinolysis. Heat completely destroyed their activity.

On the same date these experiments were made, a phenoltetrachlorphthalein test was done. The excretion was very low, being only 5 per cent. Lipase was also low.

The patient was bled again on December 16, and the above tests repeated with similar results. Fibrinolysis was complete in four hours. It was destroyed by heat at 65° C. for 30 minutes, and by adding to the plasma an equal part of normal human serum.

The fibrinogen content of this plasma was .120 gm. per 100 cc. One month later the patient came to autopsy. Unfortunately the blood was not examined at this time.

SUMMARY.—Colored male, aged 50, with ascites, occult blood in the stools, and leucocytes 6000, came to autopsy. The liver showed advanced atrophic cirrhosis. One month before death blood drawn from an arm vein clotted in normal time. The clot dissolved completely in 4 hours at 37° C. Heat at 60° C. for 30 minutes inhibited this activity. Fibrinogen content of this plasma was .120 gm. per 100 cc. The excretion of phenoltetrachlorphthalein was very low, *i. e.*, 5%. Lipase was low.

CASE IV.—W. J. K., white male, age 40. Admitted to J. H. H. November 21, 1913.

P. I. began one and one-half years ago with swelling of feet and ankles; later swelling of abdomen. In May, 1913, he vomited a large amount of dark blood. Stools contained much blood. Six days before admission patient vomited one pint of dark blood and

had bloody stools. Increasing œdema, ascites and dyspnœa. Considerable epistaxis for past year. Bleeds easily from small wounds.

P. E.: Poorly nourished; marked anemia, sallow. Scleræ slightly jaundiced. Ascites. Oedema of feet and ankles. Positive Wassermann. Improved under antisyphilitic treatment. February 20, 1914, surgical operation. Omentoplasty. Recovered from operation satisfactorily. Gradually became worse. Died May 14, 1914.

Anatomical Diagnosis.—Atrophic cirrhosis of liver; portal stasis; ascites; dilated collateral circulation; organizing thrombosis of portal vein and its radicles; chronic interstitial hyperplasia of spleen; chronic interlobular pancreatitis; pancreatic necroses; hemorrhages into the islands of Langerhans; chronic interstitial nephritis; jaundice; decreased fibrinogen; fibrinolytic ferment in blood; chronic fibrous pleuritis (right); bilateral atelectasis (lower lobes); chronic local peritonitis, perihepatitis and perisplenitis.

Body is that of a white man, greatly emaciated and extremely jaundiced. There is a scar of an old surgical incision over the right rectus extending longitudinally. The abdomen is distended and the lower border of the ribs protrudes on each side. There are no superficial abnormalities noted, except dilatation of the superficial veins over the abdomen and chest wall. There is no distinct caput medusæ but the superficial vessels generally over the abdomen and chest are enlarged. On section the subcutaneous fat is almost entirely wanting. The skin adheres pretty firmly to the bony structures. On opening the peritoneal cavity a great amount of orange colored fluid is present. The omentum is folded back and adheres to the anterior abdominal wall and the anterior portion of the liver. The liver cannot be seen. Its normal site is occupied by ascitic fluid. On passing the hand back over into this region the liver can be felt deep down as a small nodular mass which feels as if it were floating in fluid. The entire organ can almost be encompassed by the palm of the hand. The spleen is very large indeed. The surface of the small bowel is everywhere smooth, but there are many irregularly linear thickenings of the serous surface, and the wall of the gut is œdematous and thick. The vessels of the mesentery about the colon and of the omentum are greatly dilated. The omentum is adherent to the anterior surface of the liver in a few places, and the vessels are dilated. They are quite small and quite incapable of carrying on much circulation. On opening the chest wall the pleural cavity and pericardial cavity are found to contain a normal amount of fluid. The right lung is adherent to the chest wall above and to the diaphragm below by fibrous adhesions.

Liver is unusually small, presenting a uniform, advanced, atrophic cirrhosis. It weighs 810 gm. It is extremely hard and its surface is typically hobnailed—composed of nodules measuring up to 4-5 mm. in diameter, which are greenish-yellow in color, surrounded by depressed connective tissue in which are dilated venules. Just above the gall bladder is a large, depressed scar which measures 4 x 5 cm. in diameter. Beneath this scar about 1 cm. below the surface is found the main branch of the portal vein almost completely thrombosed, and as the scarring above this follows the course of the vein, it seems quite probable that thrombosis of the vein had to do with the atrophic scarring. In the scar there are no dense connective tissue bands, but small ducts, and the vessels can be seen indicating a simple atrophic scar rather than one formed with proliferative connective tissue. On section the liver has lost its normal architecture, and the cut surface is composed of greenish, orange areas of hypertrophic parenchyma. These areas measure up to 5 mm. in diameter and correspond to the nodules of the surface and are surrounded by depressed scar tissue.

Microscopically the liver is found to be composed of a net work of connective tissue bands surrounding islands of large and small liver cells. Mononuclear infiltration and bile duct proliferation are quite evident.

Blood Examination.—On November 25, 1913, liver function tests were done on this patient. Except for a rather low fibrinogen, .222 gm. per 100 cc., there was no abnormality. Active fibrinolysis was present. Blood was drawn on November 25. It clotted normally and was kept on ice over night. Next morning the clot showed little change. It was placed in the thermostat and became completely fluid in five and one-half hours.

This patient came to autopsy May 14, 1914. Blood aspirated from the heart immediately after death showed marked anemia. Clotting time was normal. Complete fibrinolysis occurred on incubating the clot for five hours at 37° C. The fibrinogen content had fallen to approximately .150 gm. per 100 cc.

SUMMARY.—White male, age 40, with ascites, œdema, jaundice, tendency to bleed, anemia and positive Wassermann, eventually came to autopsy. The liver was in a state of extreme atrophic cirrhosis. Six months before death his blood clot dissolved on incubating it 5½ hours at 37° C. Fibrinogen content of the blood at this time was .222 gm. per 100 cc. Serum lipase was normal. Phenoltetrachlorphthalein excretion was normal.

Blood drawn from heart immediately after death clotted in normal time. Its clot dissolved completely in 5 hours at 37° C. Fibrinogen content of this plasma was .150 gm. per 100 cc.

SUMMARY OF CASES.*

Case.	Date.	Fibrinogen gm. per 100 cc. blood.	Fibrinolysis complete.	Phenoltetrachlorphthalein	Blood lipase
Normal..	.....	.450	Days	30–40%	0.25
Case I...	10/ 6/13	.183	3½ hrs.	.....	....
Case II..	10/21/13	.350	16 hrs.	30%	0.28
Case II..	11/19/13	.0975	3½ hrs.	.....	....
Case III..	12/13/13	.120	4 hrs.	5%	0.19
Case IV..	11/25/13	.222	5½ hrs.	30%	0.28
Case IV..	5/14/14	.150	5 hrs.	.....	....

* Phthalein and lipase figures are from tests made by Rowntree, Marshall and Chesney.

Specimens of blood from fifty cases on the wards with various diseases have been incubated at 37° C. for several days without the clot completely digesting. Even when no aseptic precautions are taken dissolution is exceedingly slow. No case of atrophic cirrhosis has come under our observation during this study that has not shown active fibrinolysis of its blood clot.

DISCUSSION.

Herein are reported four cases of atrophic hepatic cirrhosis, the diagnoses of which have been confirmed by autopsy. In each the blood has exhibited the phenomenon of active fibrinolysis, to the extent that it dissolved its own clot completely within a few hours at body temperature. In three of the cases this property of the blood has been demonstrated sometime before death.

As to the nature of this process there can be little doubt that fibrinolysis is due to enzyme activity. All the above tests point to this. The activity is very pronounced at body temperature; it is greatly injured by heat at 60° C. and destroyed

at 65° C.; it is inhibited by normal serum. After standing two or three days a plasma gradually loses most of its activity, so that dissolution of its clot proceeds very slowly.

The clots are to all appearances normal. The thrombin factor does not seem to be at fault; for oxalate plasma clotted with normal thrombin digests its fibrin quite as rapidly as when coagulated with calcium.

These facts not only support the theory that fibrinolysis is the result of enzyme activity, but are positively opposed to the view of simple fibrin dissolution. Especially so is the fact that plasma gradually loses its power to dissolve the clot. There is nothing reasonable to support the latter hypothesis.

Whether the ferment is specific for fibrin is not evident. Certainly fibrin is attacked with much greater rapidity than egg albumin or fibrinogen. Mett's tubes showed no sensible digestion after incubating in fibrinolytic serum 10 hours at 37° C. Fibrinogen does not appear to be diminished in amount in plasma incubated 24 hours. It would be a pleasing theory to attribute to activity of the fibrinolytic ferment the low fibrinogen content encountered in these cases. Evidence, however, does not support this, and we have an adequate explanation of the low fibrinogen in the severe injury to the liver; for upon this organ fibrinogen equilibrium of the blood is principally dependent.*

We have no evidence of an absolute increase in the quantity of proteolytic enzyme in the blood of these cases. The existence of proteolytic enzymes in the blood stream, not derived from formed elements of blood, is not proven. It is known, however, that leucocytes contain such enzymes, capable of acting upon fibrin. Of the cases studied here only one showed an increase in leucocytes; the others were below normal in this respect. The proteolytic enzymes emanating from this source were presumably, therefore, not increased. There was no increase in serum lipase. The inhibition of fibrinolysis by the addition of small quantities of normal serum is against the presence of a large excess of enzyme.

With the meagre facts at hand it seems more reasonable to regard fibrinolysis as the activity of normal proteolytic ferments of the blood, operating by virtue of a diminution or absence of normal antiproteolytic substances. It would be useless to speculate upon this without further data, which we hope to gather from more extensive chemical studies of these bloods.

The cases herein studied have exhibited clinically the usual signs of advanced hepatic cirrhosis. They have all manifested a tendency toward hemorrhage; and their deaths have been such as might be in great part ascribed to what is recognized as hepatic insufficiency. Fibrinolysis has thus been associated with, (a) chronic liver injury severely impairing hepatic function, and (b) with a hemorrhagic tendency.

Its association with liver injury reminds us of the instances in which fibrinolysis has been observed under experimental conditions impairing liver function. It appeared in Jacoby's phosphorus poisoned dog. As is well known liver injury is extreme in fatal phosphorus poisoning. Nolf later observed the same phenomenon after extirpation of the liver of dogs.

We have not been able thus far to demonstrate active fibrinolysis experimentally in animals acutely poisoned with phosphorus and chloroform. A few human cases of acute liver injury with necroses have come to our observation. None has shown active fibrinolysis in the blood. Again livers severely injured by advanced chronic passive congestion, but which are apparently compensating, have not been accompanied by fibrinolysis. Its presence, then, has been associated in these cases only with a stage in chronic liver disease when the organ is severely impaired functionally.

Recently three tests for liver function have been proposed and employed by Whipple* and by Rowntree,** namely, estimation of blood fibrinogen and serum lipase, and the excretion of phenoltetrachlorphthalein in the feces. In acute liver injuries under experimental conditions these methods indicate pretty accurately the degree of the liver lesion. In chronic liver disease, cirrhosis of the liver, for example, only the fibrinogen estimation seems to offer any hope as a diagnostic and prognostic procedure. In grave hepatic injury resulting from an acute or chronic process the fibrinogen content of the blood drops, sometimes to very low amounts.

In the above cases the tetrachlorphthalein excretion was diminished in only one instance; the lipase was of no diagnostic value in any; and the fibrinogen content was low in all. In Case II the fibrinogen was only slightly diminished; the other tests were normal, while the fibrinolytic property of the blood was quite active, a month before death. This makes us hopeful that the presence of a ferment in the blood capable of completely digesting its clot may be of considerable diagnostic and prognostic value in chronic hepatic insufficiency. In four cases of cardiac decompensation with ascites we have examined, where there might have been a question of differential diagnosis from cirrhosis, the fibrinolytic ferment has not been active. Likewise it has been inactive in two cases of tuberculous peritonitis with ascites.

The association of fibrinolysis with a marked tendency toward hemorrhage is also of considerable clinical interest. In Case II there is little doubt that digestion of clots in vivo was an important factor in his bleeding. At autopsy was found between the peritoneum and abdominal wall beneath a paracentesis puncture, a hematoma as large as a hen's egg. On opening it dark fluid blood escaped. Evidently what clots formed had digested, permitting a continuous ooze. A low fibrinogen content obviously also predisposes to bleeding, by forming inefficient clots. Under experimental conditions, however, the fibrinogen must drop exceedingly low, *i. e.*, about 50 mm. per 100 cc., before one may confidently refer bleeding to this factor.

SUMMARY.

1. Specimens of blood from four cases of atrophic hepatic cirrhosis have possessed the property of completely digesting their clot within a few hours, at body temperature.

2. Normal blood will not digest its clot for days or weeks.

3. Dissolution of clot in the blood of cirrhosis cases is due to an enzyme. Its activity is destroyed by heat; inhibited by normal serum; and diminished in old plasma.

4. Fibrinogen content of the blood of each case has been below normal. Hydræmia was a constant feature. One case exhibited a low phenoltetrachlorphthalein output.

5. Digestion of clots in vivo explains in part the hemorrhagic tendency present in these patients.

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THE HIGH INCISION IN CÆSAREAN SECTION.

By W. L. Thompson, M. D., New York.

The high operation in Cæsarean section [1] possesses so many advantages over the procedure commonly in use in this country that it deserves a more wide-spread publicity, and a more general adoption than it has hitherto received.

The increasing frequency with which Cæsarean section is employed to overcome dystocia, replacing to a large extent the manoeuvres at the pelvic outlet, demands a more scientific procedure than the time-honored operation with its long, mutilating incision and undue exposure and manipulation of uterus and intestines.

The simplicity of this operation and the minimum handling of the viscera involved, commend it not only to the specialist, but to the practitioner who finds himself confronted with an obstetrical emergency. I have not hesitated to perform this operation in the tenement, assisted only by students, and have never seen any untoward effects from its performance.

The photographs accompanying this article reveal at a glance the superior cosmetic effect of the short incision, and (Figs. 1 and 3) the absence of the unsightly needle holes when subcutaneous suturing is employed.

Case 49 (Fig. 2) shows a cicatrix of 2¼ inches. The operation was done at short notice, in a tenement house where all modern conveniences were lacking. The circumstances surrounding this particular case illustrate the abundant opportunities for clinical teaching afforded by a Cæsarean operation. The patient, an elderly primapara, had been in labor for twenty-four hours, with rigid, incompletely dilated cervix, and uterine inertia. I decided to perform Cæsarean section rather than forcibly to dilate the cervix and apply forceps, an operation attended with greater morbidity (when performed in the tenement under adverse conditions) than is celiotomy. I selected, as assistants, six students from the two upper classes, and for an hour rehearsed with them every procedure in the preparation for and performance of the operation. Then to each student I assigned a definite rôle and again coached him individually in his particular duties. A set of sterile dressings for laparotomy was obtained from a surgical supply house and a woman student, who had been a trained nurse, attended to the preparation of the patient and room. During the operation, a senior student gave the anæsthetic, my assistant being a senior, while the two junior assistants, the nurse, and the man who handled the baby were from the third-year class. Although the assistants were preceptibly nervous at this, their first abdominal section, yet, by proceeding slowly with a little encouragement and coaching to each man and constant supervision of their technique, the operation proceeded uninterruptedly, with the delivery of a healthy baby and the mother in good condition. Convalescence was uninterrupted and the patient on her feet in 14 days.

[1] First described by Dr. A. B. Davis, New York City, in the Bulletin of the Society of the Lying-in Hospital of New York City, December, 1905.

Technique.

Preparation.—One-half hour before the operation, the patient is given a hypodermic injection of "Ergot Aseptic," to insure proper tonicity of the uterus.

Besides the operator and the anæsthetist, three assistants are necessary: a first assistant, one to handle instruments and dressings, and a third to take charge of the baby.

The patient should be in the dorsal position, under full surgical anæsthesia with ether or chloroform.

The abdomen is prepared in the usual manner for celiotomy (especial attention being paid to the epigastric region), covered with a laparotomy sheet, and the site of the incision covered by sterile towels pinned together so as to expose the smallest possible area of skin.

Cutaneous Incision.—The skin and underlying tissues are opened by an incision 12 cm. in length (the width of the clenched fist), *ending at the umbilicus.* The peritoneal cavity is entered in the usual manner, the fundus uteri lying directly under the wound. The uterus is usually twisted on its long axis (dextro-rotation as a rule), and it will occasionally be found advantageous to manually readjust it.

Abdominal Toilet.—The intestines are carefully walled off with three or four Miculicz pads wrung out of hot normal saline. This is an exceedingly important point, as it effectually prevents exposure and handling, and minimizes the danger of contact of the intestines with the possibly infected amniotic fluid. The first assistant now places his hands on either side of the abdomen, well back toward the flanks, to steady the uterus against the anterior abdominal wall; this manoeuvre should be continued during the emptying of the uterus and until the deep sutures are tied.

FIG. 1, CASE NO. 32.

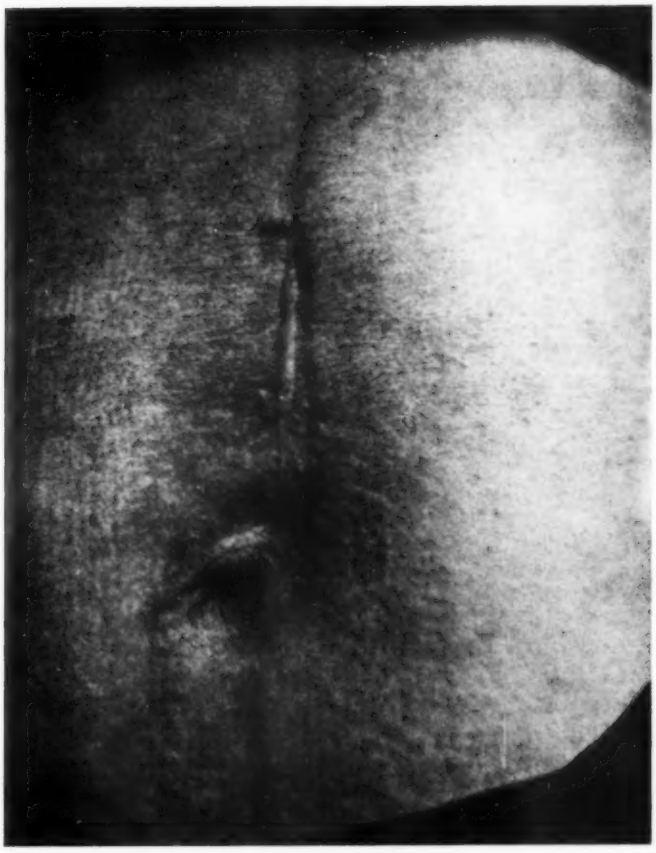


FIG. 3, CASE NO. 60.

FIG. 2, CASE NO. 49.

FIG. 4, CASE NO. 82.

FIGS. 1-4 illustrate (a) the *location* of the incision above the umbilicus; (b) its *minimal size* 12 cm. (less than 5 inches) at the time of operation, which at the end of the puerperium has contracted to 6.5 cm. (2½ inches), as in Fig. 2, Case No. 49; and (c) absence of the unsightly needle holes when subcutaneous suturing is employed.

Uterine Incision.—A median incision is now made in the uterus well up toward the fundus, and slightly longer than the abdominal opening. The incision should be made by successive strokes of the knife to avoid premature opening of the amniotic sac. As the amniotic sac bulges into the wound, the incision is best completed by scissors.

Amniotic Sac and Membranes.—The amniotic sac is not opened immediately unless the placenta lies directly under the incision, when it is cut or torn through rather than peeled away, and the uterus rapidly emptied. If the membranes alone present, the hand is swept rapidly between sac and uterus to break up any possible adhesions which might cause dangerous delay at a critical moment. The sac is punctured with knife or scissors and an adequate opening completed with the fingers.

Extraction of Child.—As the majority of presentations are cephalic, delivery of the child amounts to a breech extraction, with the head delivered by Mauriceau's manoeuvre. Should the presentation be breech, internal podalic version is performed as in a delivery *per vias naturales.* The second assistant now clamps the cord in two places, the operator cuts between them, and hands the child to the third assistant who at once removes it from the operating room to establish respiration, thus allowing the operator to give his undivided attention to the patient.

Supporting Uterus.—After it has been emptied, the uterus immediately begins to contract, and tends to slip down in the abdomen. To counteract this tendency, the pressure of the assistant's hands on the sides of the abdomen is supplemented by two double tenacula applied one to either edge of the uterine wound at its upper angle, or by hooking the fore-finger into the upper angle.

Delivery of Secundines.—Membranes, placenta and clots are now removed *en masse,* by sweeping the hand over the entire inner surface of the uterus.

Suture of Uterus.—To close the uterine wound, deep interrupted sutures of heavy chromicized gut are best. These should be inserted 1 cm. from the edge of the wound, down to, *but not through, the decidua,* passed through the opposite side, emerging at a point corresponding to the point of insertion. Beginning at the lower angle, each suture is tied as placed, and *tied tightly,* more tension being made than in suturing an ordinary wound, to allow for contraction and retraction of the uterine muscle. If this is not done, the first sutures will be found loose and unserviceable by the time the later ones are tied. Often by the time two of these deep sutures have been placed and tied, they will have acted as sufficient stimulus to cause safe contraction of the uterus and check dangerous hemorrhage, while the remaining three or four sutures usually stop all bleeding.

Hemorrhage from Uterine Wound.—Occasionally, hemorrhage from the cut uterus is alarming; muscular tone seems wanting, the whole organ a flaccid, empty sac, with blood pouring from every conceivable surface. An extremely hot wet towel applied directly to the wound, or a dry sterile towel packed hurriedly into the uterine cavity, will often hold the bleeding in check until the deep sutures have been placed; and on these sutures ultimate reliance must be placed to stop the hemorrhage, so that too much time should not be wasted in temporizing with other measures. When the blood is seen to come directly from the uterine sinuses, an attempt should be made to catch these with hemostatic forceps. If packing the uterine cavity has been resorted to, the packing should be gradually withdrawn as soon as the deep sutures are in place, and then these rapidly tied. Oftentimes this operation is performed with less loss of blood than in an ordinary labor, and without the use of an artery clamp, ligature, hot towel or other hemostatic agent from start to finish.

Superficial Uterine Sutures.—The deep sutures having been placed and tied, apposition of the uterine wound is completed by using enough interrupted sutures of fine chromicized catgut, superficially placed, to accomplish this. A running suture of fine catgut draws the peritoneum over the wound, burying the deep sutures, and minimizing the danger of adhesions between the uterine and abdominal wounds.

Closure of Abdominal Wound.—The uterine wound having been sutured, the exposed surface of the uterus is wiped clean with moist gauze pads and the abdominal packs removed. The latter will be found to have absorbed most of the blood and amniotic fluid. No attempt is made to irrigate the abdominal cavity.

While the abdominal wound is protected by a sterile towel, the surrounding skin is washed and wiped clean, the soiled towels removed, and a fresh toilet made with dry sterile dressings. In closing the wound, three sets of sutures are employed: continuous catgut for peritoneum and another for fascia, while the skin is best closed by a continuous subcutaneous suture of silver wire.

Dressing Abdominal Wound.—Sterile gauze pads are applied to the wound, and held in place by broad strips of adhesive. The vulva is covered by a suitable pad, and a T-binder applied.

POST-OPERATIVE TREATMENT.

As soon as recovery from the anæsthetic is complete, the head of the bed is raised enough to give a decided incline in order to favor drainage, and particularly to cause the uterus to sink downward toward the pelvis and away from the abdominal wound, to further minimize the danger of adhesions.

The silver wire suture may with safety be removed on the seventh to the ninth day; by grasping its free ends with hemostatic forceps and working the wire back and forth in its sinus a few times, its ease of removal is enhanced; one end is then cut off close to the skin and the remainder of the wire removed with a single quick pull.

Convalescence is as rapid and uninterrupted after this operation as the puerperium following a normal delivery. My patients have all been allowed to sit up on the tenth day, have walked on the twelfth, and have been going up and down stairs on the fourteenth with no untoward effects either upon their general well-being or the progress of involution.

There is no reason why as much deliberation should not be

exercised in the performance of this as in any other intra-abdominal procedure. At no time in the course of the operation is there need for haste, with the occasional exception of placing and tying the deep uterine sutures.

SUMMARY.

The salient features of this operation, which should give it first place among the procedures for Cæsarean section, may be briefly reviewed.

1. *Skin Incision.*—(a) Its *location*, high in the abdomen, above the umbilicus, renders it less subject to pressure than in the more convex and dependent portion; it receives better re-enforcement from the recti muscles whose edges are nearer together at this point in the parous woman, and whose support is greater as the upper attachments of these muscles are approached. Its location, furthermore, precludes all possibility of adhesion between it and the rapidly contracting and retracting uterus, whose cut surface, by the time the uterus is emptied, is far removed from the abdominal incision.

(b) Its *length*, only 12 cm. (less than 5 inches), see Fig. 4, in contrast with the usual incision of twice this length, leaves an exceedingly short cicatrix which is rendered almost inappreciable by the practice of subcutaneous suturing, as is well illustrated by the accompanying photographs.

2. *Packing off the intestines* not only keeps them out of the way during operation, but in the event of repeated vaginal examinations under uncertain aseptic precautions, prevents the possibly infected liquor amnii from inoculating the abdominal contents.

3. *Leaving the uterus in situ* has more to recommend it than is at first apparent. It is an accepted fact that sudden reduction of tension in a body-cavity tends to induce shock (from acute cerebral anemia); such an accident is scarcely possible with the uterus in situ. The common practice of delivery of the uterus and manipulation of the intestines carries with it the constant danger of infection of these structures.

4. *Uterine Incision.*—The location of the incision in the thick, muscular fundus minimizes the danger of rupture in subsequent labors, whereas the long incision in common use extends into or encroaches upon the attenuated lower uterine segment and further increases its vulnerability.

NITROGEN METABOLISM BEFORE AND AFTER SPLENECTOMY IN A CASE OF PERNICIOUS ANÆMIA.

By GEORGE R. MINOT, M. D.,
Assistant Resident Physician, The Johns Hopkins Hospital.

(From the Chemical Division of the Medical Clinic.)

Von Noorden [1] in 1894 was the first to study accurately the metabolism in pernicious anæmia. He was unable to show that protein decomposition was increased as a consequence of the anæmia, though he states that earlier observers believed this to be the case. He has found in some cases a retention of nitrogen, and Von Stejskal and Eben,[2] Strauss [3] and Bloch [4] have also found the same; it is, perhaps, due to a previous underfeeding, or associated with periods of blood regeneration, as suggested by Mohr.[5] Yet, with a rapid destruction of red blood corpuscles, there may be increased protein destruction.

Rosenquist [6] found in cases of pernicious anæmia that periods during which nitrogen excretion was increased alternated with those in which a retention occurred, but believed there tended to be a pathological decomposition of protein. Von Noorden [1] and Umber [7] have suggested that Rosenquist's methods may have been at fault in the way he calculated nitrogen in the faces, and that in anæmia functional disturbances of the alimentary canal and kidney do occur which could cause irregularities in the excretion of nitrogen and serve to explain any temporary increase or retention of nitrogen, provided the pathological deviations do not reach too high a degree. In pernicious anæmia Mohr [8] thinks that we may have a retention of nitrogen in some cases, even on a low nitrogen intake, while in others a loss of nitrogen may occur on a higher intake, and that the nitrogen exchange is not dependent on the hæmoglobin poverty, but controlled by other factors, such, possibly, as the influence of toxic substances which vary in intensity.

In severe anæmia, due to *Bothriocephalus latus*, where we have a known toxic action, as shown by Talliquist,[9] Rosenquist [6] was able to show a definite loss of nitrogen dependent on the occurrence of the worm in the body, while after its removal a retention was usually found (Bohland [10] and Vannini [11] have observed a similar condition in Anchylostoma anæmia); he thus regards the loss of nitrogen as due to a toxine and not to the anæmia, *per se*.

Cases of pernicious anæmia can be kept in nitrogen equilibrium on approximately the same small quantities of protein as normal individuals, as shown by Bernert and von Stejskal.[2]

Partition of Urinary Nitrogen—(a) Urea: Von Noorden [1] noted a "marked reduction of the per cent of urea nitrogen in some cases of pernicious anæmia." These cases were in an advanced state and with œdema. He quotes other observations with normal values. Voges [12a] found this diminution of per cent of urea nitrogen only as an exception. Podoa [12b] in 1909 believes there is a diminution of per cent of urea nitrogen in all forms of anæmia. In experimental pyrodin anæmia in dogs, Samuely,[7] in a careful metabolic study, found that as the anæmia progressed the urea per cent dropped from 86.3 per cent to 72.14 per cent.

(b) Ammonia: Von Noorden[7] and others find the ammonia normal, or a slight tendency to a relative increase in this disease.

(c) Amino-acid fraction: This fraction has in some instances been found to be increased, though there may be some doubt as to their value, since the determinations were made by older methods. Erben,[8] in one case, found the amino-acid nitrogen distinctly increased, and Samuely,[9] in his experimental anæmia, showed a definite rise which he felt might be explained by other causes than the anæmia.

The effect of removal of the spleen on nitrogen metabolism was studied by Noel Paton,[9] in 1900, on dogs in the form of urea and ammonia. He found no essential difference before and after splenectomy.

Mendel and Gibson[10] in the case of a man with enlarged spleen and secondary anæmia with ascites, following malaria, studied the metabolism after splenectomy, during which period pneumonia developed. Their conclusions are, "analytical data fail to indicate any striking variation from the normal distribution of urinary components which can be associated with the exclusion of the function of the spleen." The nitrogen intake was not studied. Monaczweski[10a] studied nitrogen metabolism in a case complicated by pneumonia seven months after splenectomy and "no noteworthy variation was seen from that in fevers."

The most valuable work is that of Umber,[5, 11] who showed in two cases of Banti's disease that there was a pathological destruction of protein and after splenectomy a normal metabolism was established. He explains this on the basis that by removing the spleen we remove the toxic cause. His work is careful in detail, and he emphasizes the necessity for all requirements of a careful balance sheet, for rough methods may not show us the nitrogen loss; moreover, pathological destruction of protein may be overlooked if we allow too high an intake of nitrogen, for then we may find a nitrogen balance as with fever cases, while on a smaller intake, yet sufficient for body needs, this would not occur. "Cases must be studied over several periods to see if there is a toxic destruction of nitrogen," he says. No essential change was found in urea per cent before or after splenectomy in the one case on which it was studied.

In March, 1913, Eppinger,[12] and also Decastello,[13] first reported the results of splenectomy in pernicious anæmia and since have reported more cases. Also Klemperer and Hirschfeld,[14] von Mosse,[15a] Huber,[15a] Port,[15] Harpole and Fox,[16] Coleman and Hartwell,[17] and Moffitt,[18] have reported similar instances, and Türk[19] discusses this procedure. In some cases great benefit has been derived from splenectomy, but of how lasting duration time alone will tell.

A case of pernicious anæmia entered The Johns Hopkins Hospital November 11, 1913. No improvement occurred and splenectomy and direct transfusion were done February 3, 1914, following which daily improvement in her condition has occurred.

A summary of the case is as follows:

Medical numbers, 31704 and 32146; surgical number, 33843. Colored (mulatto), housewife, age 35 years.

FAMILY HISTORY—Negative.

PAST HISTORY—Never ill except from measles, whooping cough and mumps as a child. Habits good.

PRESENT ILLNESS—One year ago, without previous gastro-intestinal disturbance, she began having attacks of "indigestion," which have been increasing in frequency and severity, lasting from one hour to a day, consisting of nausea and vomiting small amounts of food and "bile"-stained fluid. No pain with attacks beyond feeling of pressure of gas "around heart," relieved by belching. Attacks have no definite relation to meals, usually come when tired, also often in the morning before breakfast. Bowels have been regular, but moving twice a day. Stools have appeared normal. For six to eight weeks has had palpitation of heart, also roaring in ears. For four weeks swelling of ankles. In recent weeks has noticed that she is tired easily, and has lost weight—average with clothes 140 lbs.; now 110 lbs. Had to stop work one week ago.

PHYSICAL EXAMINATION (composite by Drs. Thayer, Boggs, Clough, Austrian, F. J. Smith, in November, and G. R. M. in January)—A well developed mulatto woman, fairly nourished, complaining of weakness, appears strikingly pale, with yellow tinge to skin and very pale mucosæ. *Scleræ* have icteroid tint. *Pupils* negative. Puffiness about eyes. *Nodes:* cervical enlarged to the size of peas, few small ones under angles of jaws; axillary, inguinals, epitrochlears not felt. *Ears, nose, tonsils, thyroid,* all negative. *Teeth:* slight pyorrhœa; otherwise negative. *Tongue:* a little raw towards base. *Throat* negative save for a few white patches over buccal mucosa. *Thorax* well formed; costal angle a little under 90°. *Lungs* negative. *Heart:* point of maximum impulse not seen; felt in fifth interspace, 8½ cm. to left of mid-sternal line; right border 3-3½ cm. from mid-sternal line in fourth interspace. *Sounds* are regular; at apex first sound muffled, followed by a soft blowing systolic murmur transmitted to axilla and well heard over body of heart, being loudest at pulmonic area, where it sounds rougher. Some months later this murmur was not heard in axilla, otherwise as before. Second pulmonic slightly accentuated.

Pulses: equal, regular, small volume, vessel walls not felt. Blood pressure, systolic 115; diastolic 60 (Tycos).

Rate per minute:

November 11-January 11	85-100
January 11-February 3	90-110
February 3-February 16	105- 90
February 16-April 29	80- 90

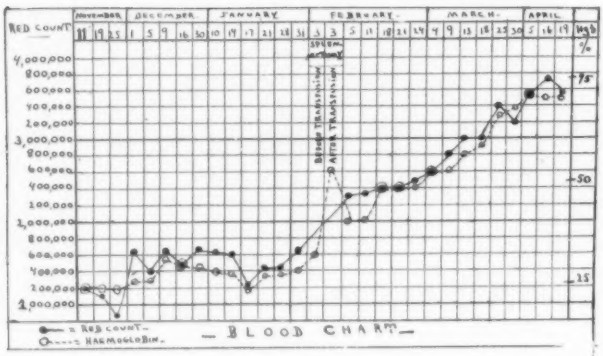
Temperature showed slight daily fluctuations:

November 11-November 29..100.0°-99.0° F.
November 29-February 3.... 98.0°-99.5° F., rarely to 100° F.
February 3-February 9.....101.0°-99.0° F.
February 9-February 16.... 99.5°-98.6° F.
February 16-March 16..... 98.0°-99.0° F.
March 16-April 29..... 98.6° F.

Abdomen soft relaxed, slight diastasis of recti; intestines or stomach may be seen to descend in region of umbilicus with inspiration. No visible peristalsis. Slightly full in right upper quadrant, generally dull tympany, no tenderness. *Liver* dulness reaches in right nipple line from fifth interspace to 2½ cm. below costal margin, and to 4 cm. above umbilicus in mid-sternal line, where edge is felt, smooth and firm. *Spleen:* edge is just felt at costal margin, firm and rounded. *Kidneys:* right palpable and prolapsed; left is just felt.

Extremities: moderate soft œdema of legs below knees with tenderness over periosteum; otherwise negative. *Reflexes* normal. *Sensation* normal.

Blood: see chart for red count and hæmoglobin. See table for white count, differential counts and blasts. (Drs. Brush, Venable, and Fleming.)



DIFFERENTIAL COUNTS.

	Nov.		Dec.	Jan.				Feb.		Mar.			Apr.
250 white cells counted.....	13	21	2	8	14	22	28	12	17	6	18	30	16
Polynuclear neutrophiles.....	52.8	44.4	59.2	66.4	62	54.8	56.8	74.4	64.4	50.3	50	53	65
Eosinophiles.....	.8	.4	.4	1.2	.8	.8	.8	1.6	.4	4.7	2.8	1	2
Mast cells.....	.4	.4	2.4	0	.4	0	0	.4	0	0	1.2	2	0
Small mononuclears.....	27.6	31.2	21.6	29.2	31.2	40.4	34	12	20.8	34.7	26	23	8
Large mononuclears.....	15.6	20.4	12.6	2.8	4	2.8	6.4	5.2	9.2	8.3	14	15	23
Transitionals.....	1.6	2.0	1.2	.4	1.6	1.2	2	5.6	5.2	2.0	4	5	2
Myelocytes.....	0	0	1.2	0	0	0	0	.8	0	0	0	1	0
Unclassified.....	1.2	1.2	2	0	0	0	0	0	0	0	2	0	0
Blasts seen in counting 250 white cells. Normo.	1	2	4	3	3	4	4	22	?	7	1	0	0
Megalo.	3	7	11	1	3	0	0	0	?	0	0	0	0
White count.....	2740	2640	.....	....	3400	2960	3960	....	7320	3800	4890	8400	10200

Nov. 11 to Jan. 28, 17 white counts made; extremes = 2300 and 4780.

Feb. 21, 6640
" 24, 7360
Mar. 9, 5640

Mar. 13, 4980
" 25, 8600
Apr. 5, 1200
" 29, 10400

Seven stained smears and several fresh specimens of blood were studied in the period before splenectomy and all showed essentially the same picture. The red cells appeared full of hæmoglobin, considerable variation in size with many large oval forms, numerous small cells, striking variation in shape, dumb-bell and sickle forms, etc. Some polychromatophilia, at times a few stippled cells. No parasites, a few nuclear particles (Jolly bodies). Plates appeared decreased. Smear of February 17 showed reds as before. Plates strikingly increased over numbers in smears before splenectomy and distinctly above normal. Nuclear particles markedly increased. On February 28 vital staining showed 8 per cent of reticulated red cells.

Smears of March 3 and 30 and April 16 always showed red cells of the type found in pernicious anæmia, but the variation in size and shape became less marked; there was no stippling, and very rarely polychromatophilia and the nuclear particles were somewhat fewer. Fragility of red cells not studied. Coagulation time in November slightly increased; in April normal.

Gastric analysis: (November) stomach of normal size, slightly prolapsed; no stasis. No free HCl in test meal. Acidity 8 per cent deficit. Total acidity 32 per cent; otherwise negative.

Stools: in November, normal except for occasional rare fatty-acid crystals. In January often soft, mushy, with abnormal amount of gas formation and acid reaction, excessive amounts of cellulose at times, not infrequently fatty-acid crystals and soap needles; otherwise negative. After splenectomy, in February and March often bulky and soft, but with less gas formation; at times a strong alkaline reaction, and at times signs of disturbance of fat absorption, becoming essentially normal in April. (Urobilin and iron not studied.)

Urine: normal to pale color, usually slightly cloudy; specific gravity 1011-1020, acid reaction; *albumin* usually absent, rarely present as a slight trace, except for about ten days following operation, when it was constant; *sugar* absent; *guaiac* negative. Several times in January a faint acetone reaction occurred, associated usually with attacks of nausea. No diacetic; bile negative. *Sediment* consisted only of vaginal debris and a few round cells.

The determination of urobilinogen by Ehrlich's paradimethylamidobenzaldehyde test was made in January some ten times, always with strictly fresh urine, and was found present, though the reaction was not strong. Two days after splenectomy there was a very intense reaction which persisted for three days, and then gradually grew less intense, disappearing by February 18 (15 days after splenectomy) and remained absent, as it did in Hüber's case.

Wassermann reaction: November (Dr. Bloomfield), negative.

Lumbar puncture: January (Drs. Miller and Levy), normal count. Wassermann, Nonne, and gold chloride tests negative.

Calmette Tuberculin test: November (Dr. Levy), 1 per cent and 5 per cent negative.

Phenolsulphonephthalein renal function test: January (Dr. Snowden), normal.

Levulose tolerance: 100 g. in January taken; none appeared in urine. Vomited following ingestion of 125 g. After splenectomy, in March, 100 g., vomited twice—75 g. taken and none appeared in urine.

Phenoltetrachlorphthalein test for liver function: (Drs. Rowntree and Chesney), January 14, 27 per cent excreted in fæces.

Under treatment of rest for three months in bed, with proper diet and arsenic, the patient showed no improvement, nor did 375 cc. of defibrinated blood given intravenously on November 30 cause any real change in the picture. On January 13 2.5 cc. of whole blood was given without effect.

The case being considered one favorable for splenectomy, she was transferred to the surgical service February 3, on which day Dr. Finney operated. Immediately afterwards Dr. McClure gave a direct transfusion from her husband, who belonged to the same iso-agglutinin group (tested by Dr. Moss).

The patient made a good surgical recovery and held the blood. Though there was no severe hemolytic crisis, her conjunctivæ became yellower and the urobilinogen increased in the urine, lasting for a few days after the operation. She was transferred back to the medical service with all wounds healed, February 15.

Physical examination (Drs. Levy and Clough) at this time revealed nothing new, save that there was less icteroid tint to scleræ than in November. In March the liver border was thought not to be as low as formerly.

Following the operation the patient began to improve daily. She gained weight, the "indigestion" left, and many little aches disappeared as the blood count rose. She was allowed to get up the middle of March and left the hospital April 29, with no yellow tinge to skin or scleræ. At this time, although the blood picture was greatly improved, yet it was still that of a primary anæmia. A letter to Dr. Thayer in June said her condition was as good as when she left the hospital.

To study the nitrogen metabolism, the patient was kept in bed during all observations and given a measured and weighed diet of proteid, fat and carbohydrate in the proportion of about 20, 30 and 50 per cent respectively. Calories were estimated by tables. Her calorie intake was low, 25 per kilo, which is but barely enough for a patient in bed; nitrogen intake was sufficient. There was considerable difficulty in per-

suading her to eat, especially the same amount each day, which has resulted in an undesirable variation of the nitrogen intake. In all instances for twenty-four hours before the days on which the examinations were made the patient was kept on as nearly the same diet as on the days on which the determinations were made, the third day having the greatest variation. Consecutive days were not always obtained, owing to errors of collections. Other days, besides those tabulated, were studied, which tended to show the same results, but are not recorded, as errors in collections or determinations were feared.

The total nitrogen in the food, urine, and stool (dried and powdered) was determined by the Kjeldahl method, two parallel determinations on each specimen, as was also done in determining the ammonia by Folin's older method.[20] The urea was determined by Marshall's method,[21] three samples being tested each time. The results are tabulated.

verse the result. The patient lost weight in the period before splenectomy and was gaining weight in the period after, while the average intake of nitrogen was slightly greater (between test days she was actually eating more than before splenectomy), it does not seem enough to account for the retention.* Of course, it would have been desirable to have determined on high and low intakes of nitrogen what the balance would have been, but this could not be done.

Various possibilities occur to one as to why such a difference might exist; among others, it might be due to the fact, as in Umber's case, that we have removed a toxic cause, or that which allows a toxic cause to exercise its full function; or the loss might be associated with the slight fever, in turn perhaps associated with circulating toxic substances present before splenectomy but not found afterwards at the time when the tests were made; or that we found the patient in one of Rosenquist's periods of nitrogen loss and afterwards in a period of retention.

NITROGEN METABOLISM DATA.

(A) BEFORE SPLENECTOMY.

Date.	No. of day.	Calories per Kilo.	Nitrogen intake.	Total Nitrogen output.	Amount of urine.	Nitrogen in urine.	Nitrogen in stool.	Urea in urine.	Urea Nitrogen.	Urea Nitrogen.	Ammonia in urine.	Ammonia Nitrogen.	Ammonia Nitrogen.	Albumin in urine.	Weight of patient.
			gm.	gm.	cc.	gm.	gm.	gm.	gm.	%	gm.	gm.	%		
January 22–23	1	25	5.0	6.68	800	6.08	.60	7.2	3.36	54.9	.58	.47	7.7	0	Nov. 16—108 lbs.
" 23–24	2	—	—	12.07*	1375	11.24*	.83*	12.04	5.60	50.0	.86	.70	6.1	0	Jan. 21—105 lbs.
" 24–25	3	31	6.74	6.91	1125	6.14	.77	7.69	3.58	58.1	.45	.37	6.0	0	
" 28–29	4	25	12.9	14.60	2180	14.12	.47	10.46	4.88	34.5	.37	.30+	2.1	Very slight.	
" 30–31	5	25	8.75	9.25	1860	8.37	.50	13.40	6.20	74.0	.43	.35	4.1	0	Jan. 30—102 lbs.
Average	—	—	8.34	9.03	1468	8.67	.56	10.15	4.72	54.3	.53	.43+	5.2		

Average Daily Amount of Nitrogen *Lost:* .78 gm.

*Not in average.

(B) AFTER SPLENECTOMY.

Splenectomy Feb. 3d.															
February 18–19	6	25	9.35	7.73	1680	6.49	1.24	10.18	4.75	73.1	.4+	.35+	5.4	0	Feb. 16—101½ lbs.
" 19–20	7	25	9.49	7.50	1100	5.85	1.64	10.23	4.77	81.5	.4—	.35	5.9	0	
March 5–6	8	25	8.75	8.50	1775	7.54	.96	12.80	6.00	78.8	.5	.41+	5.4	0	
" 6–7	9	25	8.59	8.75	1970	7.55	1.20	13.47	6.28	83.2	.58	.47	6.2	0	
" 19–20	10	25	8.94	9.96	1900	9.04	.91	15.85	7.39	81.9	.62	.51	5.6	0	
" 21–22	11	25	9.42	8.51	1725	7.70	.80	13.10	6.10	79.2	.59	.48	6.2	0	Mar. 22—108 lbs. ?
Average	—	—	9.09	8.49	1691+	7.36	1.12	12.60	5.88	79.6	.51+	.42	5.7		April 6—112 lbs.

Average Daily Amount of Nitrogen *Retained:* .6 gm.

The total nitrogen output before splenectomy fluctuated daily; this was due to the unfortunate variation in the intake. On the fourth day the high output in urine (14.12 g.) may be associated with the presence of a slight trace of albumin. However, an average of four days, on which the output and intake were determined, tends to show a slight daily loss of nitrogen, .78 g., and each day, except No. 2, in itself shows a loss as much as in Umber's cases of Banti's disease. Yet, with more observations and a more equal amount each day, it might be quite possible to show a gain instead of a loss.

Following splenectomy the daily intake and output over six days (in three periods of two days each about two weeks apart) are more nearly equal each day (the patient being better and more willing to eat as desired). Here the average shows .6 g. of nitrogen retained per day. This is not a very essential change; for the average of a few days, like No. 5, where there was a loss of 1.2 g., or No. 8, where a loss of .25 g. might reverse the result. However this may be, coincident with clinical improvement there appeared a more favorable nitrogen balance.

A low per cent of urea nitrogen in some cases of pernicious anæmia has been spoken of in the literature. In this case on the days before splenectomy it averaged 54.3 per cent, lower than would be expected to correspond with the total amount of nitrogen in the urine, day 4 showing but 34.5 per cent, seems almost as if there must be an error. Again day 5, with output of 8.37 g. of nitrogen, shows 74 per cent urea nitrogen, in itself low, but does not seem to vary in the proportion one would expect, when day 2, with an output of 11.24 g. nitrogen, shows but 50 per cent as urea nitrogen.

On the six days studied after splenectomy the urea per cent averaged 79.6, on an average output of 7.36 g. of nitrogen in

* The stool contained less nitrogen before than after splenectomy; the significance if any, is questionable.

the urine, which is less than the average output before splenectomy (9.19 g., which includes all five days), when the per cent of urea was much lower. Here one is surprised to find a figure as high as 81.5 per cent on day 7, when but 5.85 g. of nitrogen were found in the urine, the other days being about normal. It is possible that the low urea per cent before splenectomy was associated with deranged liver function, as is suggested also by the phenoltetrachlorphthalein test, while after removal of the spleen some change occurred (disappearance of some circulating toxic substance?) which allowed the liver to functionate more normally; or again, following Padoa's work, it might be due to anæmia, *per se*, the change associated with improvement.

No changes or abnormalities were noted in the ammonia of the urine.

Richet,[22] as a result of his studies of splenectomized dogs on which for a period of two years their daily intake of food and their body weight were studied, concludes that the spleen exercises a function on nutrition, for after splenectomy the dogs ate more food to keep up their body weight. This might suggest that after splenectomy a loss of nitrogen would occur on the same diet that before splenectomy allowed nitrogenous equilibrium. As far as one can judge from the data obtained in the case of pernicious anæmia this would not hold true. We are, however, dealing here with a pathological condition of man and not with normal dogs.

It does not seem justifiable to draw conclusions on the normal function of the spleen from the study of cases where this organ has suffered pathological changes.

To conclude, in a case of pernicious anæmia showing no improvement for three months, splenectomy was performed, after which the patient's general condition and blood picture improved. Coincident with this improvement the urobilinogen disappeared from the urine, a more favorable nitrogen balance occurred, and a rise in per cent of urea nitrogen, which previously had been below normal.

It is a great pleasure to acknowledge my indebtedness to Dr. Thayer, whose suggestion it was that some metabolic studies be made in this case, and under whose supervision this work was performed; to Dr. J. H. King for many helpful suggestions in carrying out the work, and to Dr. R. R. Snowden for aid in technic.

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NOTES ON NEW BOOKS.

Materia Medica, Pharmacology and Therapeutics. Prescription Writing. By WALTER A. BASTEDO, Ph. G., M. D., etc. Illustrated. (*Philadelphia and London: W. B. Saunders Company, 1913.*)

This book of 559 pages is an adaptation for the most part of lectures delivered at Columbia University by Dr. Bastedo. It is inclined to be elementary in character and is essentially a text-book on pharmacology for the students.

In the preface the author states that he has not attempted to compile extensive bibliographies but he refers to certain works on pharmacology and physiology which he has freely consulted. The book does not impress one as the outgrowth of first-hand knowledge gained from personal experimental work or from thorough familiarity with original sources. The author is inclined to be dogmatic and has not always exercised good critical judgment, accepting much as proven where reasonable room for doubt still exists. Certain theories are accepted at times as the true explanation of certain phenomena and not simply as theories. As a pharmacology the book does not fill a definite need.

Infections of the Hand. By ALLEN B. KANAVEL, M. D. Second Edition. Thoroughly Revised. Illustrated. (*Philadelphia and New York: Lea & Febiger, 1914.*)

The importance of this work to all surgeons was noted in its first appearance in the BULLETIN, and it is unnecessary to do more than call attention to the second edition, which has given the author an opportunity to add to its value by fuller detail along certain lines. Surgical internes and students, working in dispensaries, could not do better than study this book thoroughly, for in infections of the hand much bungling surgery is constantly exhibited.

Time and the Timeless—Songs of Shadow and of Hope. By a PHYSICIAN. 2/6. (*London: Henry J. Glaisher, 1914.*)

While these verses from a physician cannot be ranked with those of Holmes or Weir Mitchell, yet this small, neatly printed volume deserves to be placed on the same shelf as those of these authors, for the poems are of more than usual merit. They exhibit deep religious feeling and while they are of shadow, the

shadow is lit by hope. They are the work of a serious thinking man of high ideals, and whereas the author's thoughts are not rare, his expression is pleasing and will be attractive to many by its simplicity and sincerity. The introductory verses are symbolic of the author's attitude of mind.

A few stray flowers of song
Found in my garden. Call them only weeds,
But Nature sowed the seeds,
And unto her do they belong.

She fed them, while men slept, with smiles and tears
And they have flourished there.
I would it were an offering more fair,
Fitter for human needs,
Mixed with the sterner harvests of the years.

A verse to stimulate us all runs as follows:

We act ourselves:
And in the actions, as in a mirror, shine
Our features line by line,
And if we bend our wills to better deeds,
We hope to trace
In them a nobler face.

Black's Medical Dictionary. By JOHN D. COMRIE, M. A., B. Sc., M. D., F. R. C. P., Edinburgh, Lecturer on History of Medicine, University of Edinburgh; Lecturer on Clinical Medicine, University of Edinburgh, etc. Fifth edition, completing thirty thousand, containing 431 illustrations in the text, etc., and 12 plates in color. (*New York: The Macmillan Company, Publishers, 1914.*)

This book of nearly 860 pages is not so much a medical dictionary in the opinion of the reviewer as a dictionary of medicine, modelled somewhat after the manner of Quain's dictionary. It is not compiled so much to assist the medical student to acquaint himself with the meaning of the words which he finds in his text-book and journals as to render it possible for "district nurses, teachers, clergymen and professional men generally, ship-captains, colonists, business men and others" to know something of medicine and possibly, although it is not so stated, to practice the healing art in an emergency. It contains special directions for the treatment of accidents, for methods of recovery from drowning; for the treatment of fits and convulsions; for poisoning; for unconsciousness whether from alcoholism, apoplexy, opium poisoning or uræmia; and for the preservation of health by exercise, diet, ventilation and general sanitation.

It presents also the symptoms of disease, gives directions as to nursing and the management of children. It further treats of the action of medicine and of physiological matters such as circulation, perspiration, respiration, temperature, pain, sleep and vision.

The work is carefully and painstakingly written and seems fairly well abreast of modern discoveries in medicine. At the same time a glance at its pages gives one an impression that it is too much a treatise on practice and yet not specific enough to be of the highest utility. To the ship-captain and colonist it may be of real service, in default of any other medical aid; to the clergyman or amateur physician where skilled medical attendance is available, it presents too much information which may prove a stumbling block and delay adequate treatment.

The colored illustrations are well done. By a curious lack of consistency, colored pictures of chicken-pox, German measles, measles and scarlet fever are presented but there is no representation of small-pox, which is equally apt to break out in remote regions and give rise to more serious epidemics.

Sanitary topics such as water supply, ventilation, disinfection, water-closets, and sewage removal are very well written. The book as indicated above has a limited range of usefulness and will evidently fill a recognized want in isolated communities.

Diseases of Women: Medical and Surgical Gynecology. By CHARLES A. L. REED, M. D. Illustrated. $6. (*New York and London: D. Appleton & Co., 1913.*)

In this octavo volume of 872 pages of text—minus a very considerable amount of space allotted to illustrations—the author undertakes to discuss not only those topics usually treated in books of this size on gynecology, but also surgery of the urinary system, rectum and breast, as well as certain major obstetrical operations. Furthermore, the work is offered as fulfilling the requirements of a text-book for undergraduate students, a teacher's reference book and a guide, both medical and surgical, for the practitioner.

The inevitable result is, of course, the production of an incomplete and epitomized presentation of the various subjects treated.

But the fact that the book falls short of its author's aim does not seriously detract from the very considerable degree of merit it possesses. Indeed, it reflects distinct credit upon the writer since it contains a wealth of accurate information which has been wisely selected, logically arranged and tersely presented with clearness and force. The subject matter is divided into sections on malformations, injuries, displacements, foreign bodies, infections, neoplasms, trophic changes, obstetrical operations and menstruation; the subdivisions being made on an anastomic basis.

It is to be deplored that no mention is made of certain live and important gynecological subjects. Notably, vaccines in the diagnosis and treatment of gonorrhœa; the Roentgen and radium treatment of neoplasms and other gynecological affections; and the recent complete revision of our knowledge and views regarding menstruation following upon the work of Hitschmann and Adler.

On the whole the author's advice is sound and in accord with present-day teaching, but the radical operative treatment advocated in cases of puerperal infection would not be generally endorsed either in this country or Germany.

The numerous illustrations are for the most part sketchy and often semi-diagrammatic, but are of material aid in conveying the author's ideas.

Some American Medical Botanists, Commemorated in our Botanical Nomenclature. By HOWARD A. KELLY, M. D., LL.D. Delivered as a lecture before the Medical Historical Society of Chicago, 1910, and before the University of Nebraska, 1913. (*Troy, New York: The Southworth Company, Publishers, 1914.*)

This tribute by a medical botanist to men who have given their names, talents and energy to the science of Botany is graceful and timely. The volume contains more or less complete sketches of the lives and attainments of thirty medical men. A large number of them were immigrants to America in pioneer days; such men as Michel S. Sarrazin, a native of France who emigrated to Quebec at the age of 26 years and gave his name to the beautiful pitcher plant, the *Sarracenia purpurea;* John Mitchell, a native of England but a resident of Virginia, from whom the familiar checkerberry, *Mitchella repens*, received its name; Cadwalader Colden, a native of Scotland but a resident of New York later, where he was a physician, Surveyor-General of the State and Lieutenant-Governor and ultimately a botanist, a friend of Linnaeus, who named after him a new genus of plants known as *Coldenia;* John Clayton, of England, who came to Virginia as a youth and from whom *Claytonia Virginia* or *Spring Beauty* received its botanical name; others like John Bartram, Alexander Garden, Adam Kuhn, Moses Marshall, Caspar Wistar and Benjamin Barton were descended from recent immigrants and enriched the new science by their enthusiastic labors; while others like David Hosack, Asa Gray, John Torrey and Charles W. Short

seemed more nearly products of the soil. The life story of all is well told but proves quite too brief and the reader laments that concerning many of them such meagre facts are alone available. There are interesting stories of daring adventures in the wilds of a new country in search of new plants and thrilling tales of quests to establish the correctness of the discoveries of other botanists. No one can read without a thrill of the quest of Gray and others in search of *Shortia*, lost for ninety-eight years and finally rediscovered in 1886 near the exact spot where it was first discovered by Michaux!

The book is profusely illustrated by excellent reproductions of old prints and is beautifully printed. The original lecture referred to upon the title page is contained in a scholarly Introduction which contains much interesting information as to the names of plants ancient and modern. The book is most creditable to the author, who has achieved deserved success in many other fields.

Flies in Relation to Disease—Non-Bloodsucking Flies. By G. S. Graham-Smith, M. D., University Lecturer in Hygiene, Cambridge. (*Cambridge: At the University Press, 1913.*)

This volume is one of the Cambridge Public Health Series, prepared under the editorship of Dr. G. S. Graham-Smith and Mr. J. E. Purvis. It is a useful and timely publication at a time when the agency of flies in conveying disease is beginning to be appreciated in every country. The work contains chapters on the life history of the fly, his anatomy, habits, and methods of carrying and distributing bacteria, etc., which are clear and admirably illustrated. These are followed by chapters on the relation of flies to specific diseases like typhoid fever, epidemic or summer diarrhœas, cholera, tuberculosis, anthrax, and bacterial diseases like diphtheria, ophthalmia, plague and staphylococcal infections. It is of interest to note, as indicating the judicial character of the work, that the author does not seem to regard it as proven that the housefly is to any great extent a carrier of the typhoid bacillus under the ordinary conditions of city life, but inclines to the belief that he may play an important part in the direct transmission of this disease from infected typhoid patients to healthy persons in the unsanitary conditions of camp life. He believes, however, that the whole subject needs to be restudied and his attitude of mind suggests the suspicion that the domestic fly has been unduly condemned. He points out the fact that so-called typhoid carriers are much more worthy to be censured for the spread of the disease. The work will be of great value to sanitarians and health officers.

Treatment of Neurasthenia. By Dr. Paul Hartenberg. Translated by Ernest Playfair, M. B., M. R. C. P. (*Edinburgh, Glasgow and London: Henry Frowde and Hodder & Stoughton, Publishers, 1914.*)

This admirable little book of 283 pages is the sequel of a previous work by the author, "The Psychology of Neurasthenia"; it relates to treatment and cure. As the work represents the results of one who has spent many years in the treatment of this class of invalids, it is of value to all who attempt to treat one of the most intractable and discouraging of functional diseases.

The author's definition of neurasthenia follows closely the common idea that it is a state of simple depression of the nervous system and is quite as satisfactory as Polonius' definition of madness—and no more illuminating. What produces the condition, whether an exhaustion of the nerve cells or an intoxication, he does not know. He finds a condition and has no adequate theory to explain it satisfactorily. He regards it a physical and material affection, but does not explain why. He asserts, however, that it is not purely imaginary or fictitious.

To treat this condition, its causes must be discovered and eliminated. The chapters on treatment are full and give detailed methods to be pursued. He shows in what manner every neurasthenic should be studied and gives rules for both general treatment and special treatment of the individual predisposition, the exciting causes, such as dyspeptic symptoms, organic affections like gout, diabetes, arterio-sclerosis, infectious diseases like typhoid fever or influenza, dilated stomach, overwork or emotive causes. In the treatment of the fundamental asthenia he uses principally the preparations of strychnia by hypodermic injections, although a variety of other nerve tonics is mentioned. The mental state must be met by the direct personal influence of the physician, whose duty it is to give encouragement and mental stimulation to those who have lost all power of self-direction. He also believes that the application of general electricity is of service.

His methods are carefully thought out and will prove of great service to physicians who see early cases of neurasthenia in general practice. It is not an epoch-making work, but none the less a useful one.

Progressive Medicine. A Quarterly Digest of Advances, Discoveries and Improvements in the Medical and Surgical Sciences. Edited by Hobart Amory Hare, Professor of Therapeutics, etc., in the Jefferson Medical College, Philadelphia, assisted by Leighton F. Appleman, M. D., Instructor in Therapeutics, Jefferson Medical College. Volume II, June, 1914. Hernia Surgery of the Abdomen, Exclusive of Hernia, Gynecology, Diseases of the Blood, Diathetic and Metabolic Diseases, Diseases of the Spleen, Thyroid Gland, Nutrition and the Sympathetic System, Ophthalmology. The authors of the sections on Hernia and Surgery of the Abdomen are Drs. W. B. Coley and John C. A. Gerster, of New York; on Gynecology, Dr. John G. Clark of Philadelphia; on Diseases of the Blood of the Ductless Glands of the Sympathetic System and Diathetic and Metabolic Diseases, Dr. Alfred Stengel; and on Ophthalmology, Dr. Edward Jackson. (*Philadelphia and New York: Lea & Febiger, 1914.*)

The articles are in the nature of analytical and critical reviews and they contain the results of the latest work. They are too long and detailed for a fuller review, but are none the less deserving of the careful reading of physicians who desire to keep themselves informed of the progress of medicine and surgery. The series has been in progress for many years and its value is shown by the liberal patronage which it has secured.

Life and Letters of Nathan Smith, M. B., M. D. By Emily A. Smith. With an introduction by William H. Welch, M. D., LL.D. (*New Haven: Yale University Press, 1914.*)

This book has a double interest because it not only is a fitting contribution to the celebration of the Centennial Anniversary of the "Medical Institution of Yale College," of which Nathan Smith was an organizer and chief ornament, but also by reason of his profound influence upon medical education in New England and his connection with Jefferson Medical College in Philadelphia and the University of Maryland in Baltimore, through his son, Prof. Nathan R. Smith, who filled so large a place in the medical annals of Maryland, and indirectly through his grandson, Dr. Alan P. Smith, associated with the University of Maryland as a teacher and later as a trustee of The Johns Hopkins Hospital and of The Johns Hopkins University during the critical period of the foundation and organization of these institutions, which have influenced widely both general and medical education.

It has been a labor of love on the part of Mrs. Alan P. Smith to make a substantial addition to our knowledge of Nathan Smith by the publication of his interesting and hitherto unknown letters to Dr. Shattuck, of Boston, and of extracts from his lectures and writings. She has also given a charming account of early pioneer

conditions in New England during the last quarter of the eighteenth century, and of the struggles and privations which accompanied early efforts to establish medical schools.

On the personal side, to quote the words of Dr. Welch: "We catch intimate glimpses of the active-minded lad upon the frontier, of the student at home and abroad getting, in spite of great difficulties, a good medical training; of the lover 'transported with joy and expectation'; of the devoted husband and father solicitous for the education of his sons; of the busy physician and surgeon 'bandied about from one part of the country to the other,' treating fevers, couching for cataract, cutting for stone, excising tumors, and embarrassed most of the time, as is the way of doctors, from failure or inability to collect his fees, small as they were; of the founder of medical schools and the professor filling, and filling well, all the chairs in the medical curriculum, from all accounts a great teacher—and withal deserving President Woolsey's characterization of him as 'the most delightful, unselfish and kind-hearted man I ever knew and we children loved him.'"

He was indeed a great man and his characteristics are charmingly brought out in this all too-brief biography. The book is finely printed and well-illustrated by reproductions of photographs and facsimiles of original documents. It reflects great credit upon the publishers and upon the author.

The growing interest in historical medical topics lends the hope that it may be widely read and appreciated.

Auricular Flutter. By WILLIAM T. RITCHIE. (*Edinburgh and London: W. Green & Son, 1914.*)

The study of cardiac disease, especially the analysis of the arrhythmias has been advanced so rapidly by the use of graphic methods of registration, that most text books on the subject do not include all of them. In this small volume of one hundred and thirty pages the author has given an excellent résumé of a type of cardiac irregularity, only recently differentiated. While the experiments and clinical observations along this line are of great importance in developing the knowledge of the different forms of tachycardia and arrhythmia of the heart, so many of the fundamental points of auricular flutter are as yet unsettled, that any book written upon the subject may still be somewhat unsatisfactory.

The first chapter of the book is devoted to an excellent summing up of the anatomy and the normal and pathological physiology of the heart as shown by the various means of registration. This short review covers most the elementary features of cardiac irregularities and prepares the reader for the more complex subject that follows. In the remainder of the volume the condition termed auricular flutter is discussed in detail, both from the clinical and experimental standpoint. It is well supplemented by numerous curves and the histories of all such cases reported in the literature. The bibliography is very complete and the book as a whole can be commended as an excellent means by which one may become acquainted with the newer details of cardiac disease.

Tuberculosis: Its Cure and Prevention. (*A book for laymen.*) By EDWARD O. OTIS, M. D. (*New York: Thomas Y. Crowell Company, 1914.*)

A very satisfactory presentation of the important facts about the cause, prevention and cure of tuberculosis. The book is clearly and agreeably written. Though designed for laymen, it contains important material with which every physician should be acquainted. Medical students will profit by it.

Ten Sex Talks to Boys Ten Years and Over. By I. D. STEINHARDT, M. D. Price $1. (*Philadelphia and London: J. B. Lippincott Company, 1914.*)

Most physicians with some experience of the difficulties of the period of development will agree that much unnecessary distress and nervousness is caused by the conventional attitude of silence with regard to the vital topics discussed in this book. Juvenile curiosity is officially ignored, the topic of sex is taboo; the resultant compromises are often disastrous. This situation forms an organic part of the structure of modern social thought and its modification can only be a matter of slow evolution.

The author pays little attention to the complexities of the question; to him the solution appears to be to place before the developing youth a simple statement of the facts of the sexual life. As to his formulation of the facts little exception need be taken; the tone of the book is, however, essentially hortatory, and reminiscent of the prohibitionist, and one may doubt the wisdom of making the flesh of the youthful auditor creep by such discussions as those on syphilis, gonorrhœa and masturbation. If the author has not struck the right key-note, at least he is in earnest, and there may be situations where the best available solution is to place such a book in the hands of a boy of ten years or over.

Feeble Mindedness: Its Causes and Consequences. By HENRY HERBERT GODDARD, Ph. D. Pp. XII + 599. Price $4.00. (*New York: The Macmillan Company, 1914.*)

In this volume the author publishes the very valuable material which he has been accumulating at the Training School at Vineland, N. J. In the opening part he discusses the general problems of feeble-mindedness, and outlines his methods of investigation. He then presents in a brief form the case histories of 327 cases of feeble-mindedness. In the report and discussion of these cases emphasis is laid on the family history, while the clinical history of the individual is of the most meager description. This no doubt is partly due to the nature of the material, but it also represents the attitude of the author towards the whole question. In the absence of adequate clinical data a solution of the individual case will never be found by shaking the family tree, however vigorously, and the accumulation of more material is not so much required as the study of cases whose personal histories have been worked over much more intensively. In the presence of a neuropathic ancestry Goddard pays little attention to a positive Wassermann reaction; he discusses in a general statistical way questions which are only to be answered by accurate anamneses and clinical examination. After presenting his case histories the author discusses a variety of questions. He tends to the opinion that the inheritance of feeble-mindedness obeys the Mendelian law and that feeble-mindedness behaves as a unit character.

In the concluding chapters various practical aspects of the problem of feeble-mindedness are discussed and its relations to pauperism, alcoholism, prostitution and crime are emphasized. Goddard admits that the pressure of the popular demand has caused him to publish his material in a form which is far from complete and it is to be regretted that the author's critical judgment did not withstand this presure.

The book as a whole is extremely interesting and the material being presented in a somewhat popular form it may be considered a good introduction to the problems presented by the feeble-minded.

Archives of Neurology and Psychiatry, from The Pathological Laboratory, London County Asylums. Edited by F. W. MOTT, M. D., F. R. S., F. R. C. P. Vol. VI. (*London: John Bale, Sons & Danielsson, Ltd., 1914.*)

It is always a great pleasure to welcome a new volume of the Archives of Neurology and Psychiatry. The present volume may not be quite so well-balanced as some of its predecessors, but it contains many interesting and important papers.

In discussing "The Nature of the Condition termed Parasyphilis," Dr. Mott reviews the evidence which led up to the idea that general paralysis was definitely due to the syphilitic organism, and following the researches of Noguchi and Moore he has been able to demonstrate the specific organism in sixty-six per cent of his cases.

In another paper on the occurrence of general paralysis in the London County Asylums, it was found that fifteen per cent of the male admissions were suffering from that disease. On account of such a state of affairs, a strong plea is put forward for the prevention of the cause of the disease and to show how frequently the nervous system may be affected, the following appalling statistics are recorded:

In Vienna out of 4134 officers infected with syphilis 198 subsequently suffered from general paralysis, 116 developed locomotor ataxia, and 134 developed cerebro-spinal syphilis.

Candler and Mann contribute an article on "The Wassermann Reaction in the Diagnosis of Mental Disorders." This work has been most carefully done and their results correspond closely to those obtained by other workers. They have, however, in addition carried out an interesting series of tests on the cerebro-spinal fluid and blood serum obtained post-mortem and conclude that "On any case in which a clear cerebro-spinal fluid or a serum showing no hæmolysis can be obtained post-mortem, the Wassermann test on these fluids may be taken with reliance."

Within recent years Dr. Mott had interested himself greatly in the relationship of heredity to insanity and three valuable contributions to the topic are made by Drs. Mott, Wilson White, and Wooten.

Dr. Edgar Schuster has a paper on "Hereditary Resemblance in the Fissures of the Cerebral Hemispheres." A most careful description is given of the brains of a mother and daughter, and of two brothers. He also contributes a description of the convolutional pattern of the brain of a Malay.

Comparative neuro-biology is represented by a most painstaking and careful paper by Fortuyn on the cortical cell lamination of the hemispheres of some rodents. Most excellent photographs and diagrams accompany these papers.

In the appendix a series of papers is incorporated which have been previously published. Of these special mention may be made of Dr. Mott's excellent paper on "The Relation of Head Injury to Nervous and Mental Disease," and an interesting account of fatal pellegra in two English boys in which the histo-pathological findings of one case are compared with the histological changes in the nervous system found in a case of pellagra dying in the Abassieh Asylum, Cairo.

There are several other interesting communications, but the above may serve to give some idea of the scope of the present volume.

Spectrum Analysis Applied to Biology and Medicine. By C. A. Macmunn. Pp. 110. Price $1.75, Net. (*Longmans, Green & Co., 1914.*)

This small volume is an epitome of the author's researches on the spectrum analysis of the pigments, a book written between attacks of illness and based on work which was conducted after the routine activities of the day were done. Obviously the book will find favor chiefly in the hands of those trained in the difficult science of spectrum analysis and those who use this in the biological study of the pigments such as are found in the animal and vegetable kingdoms. A short introductory chapter deals with the historical side of the spectrum, the instruments used, the nature and significance of the Frauenhofer lines and the various kinds of spectra. The chapter on the "General Chromatology of Plants and Animals" is extremely interesting and includes some of the chemistry of the pigments in general. Chlorophyl, hemaglobin and its derivatives are well discussed and their characteristic spectra given in charts. The section on "Quantitative Spectrum Analysis" can only be understood by the mathematician and physicist. A useful bibliography finishes the work. The book is simply written, free from all garnishes and will doubtless prove stimulating to those who work in this highly specialized field of physical biology. Moreover it will always serve as a ready guide for those about to enter upon this particular type of research.

On Dreams. By Prof. Dr. Sigmund Freud. Authorized English Translation by M. D. Eder. With an Introduction by W. Leslie Mackenzie. $1.00. (*New York: Rebman Company.*)

Dr. Mackenzie in his introduction to this little volume hits the nail on the head when he criticies the fact that most text-books on insanity do not attempt to show how mental diseases have their roots in the mental processes of the normal mind, but usually content themselves with a purely descriptive and symptomatological statement regarding the disease process. Freud's genius in elaborating the theory of psychanalysis and of dream interpretation has happily changed that state of affairs, and has instilled new life into the study of the neuroses and psychoses.

In this short essay of 106 pages Freud lucidly and clearly sets forth his ideas in regard to the interpretation of dreams.

The book in no way replaces Freud's more elaborate work, "Die Traumdeutung," but it may serve as a useful introduction and stimulus to the larger and more comprehensive work.

Surgery: Its Principles and Practice. For Students and Practitioners. By Astley Paston Cooper Ashhurst, A. B., M. D., F. A. C. S. Instructor in Surgery in the University of Pennsylvania, Associate Surgeon to the Episcopal Hospital, and Assistant Surgeon to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases. With 7 Colored Plates and 1032 Illustrations in the Text, Mostly Original. (*Philadelphia and New York: Lea & Febiger, 1914.*)

The book is divided into three main sections: General Surgery, Systemic Surgery, Regional Surgery. In the preface the author says: Emphasis is placed on the underlying principles and pathogenesis, diagnosis and indications for treatment. The more important operations are described in detail. The specialities of the eye, the ear, the nose, and the throat are not included. Genito-urinary surgery, gynecology and orthopedics are discussed only so far as they come within the province of the general surgeon. To economize space the bibliographical references are omitted, but the dates of authoritative contributions are indicated and in this way the original references may easily be found. The illustrations are for the most part new, and are well selected. The arrangement of the chapters is good and the text is well written. It might be mentioned that some of the subjects seem to be considered too briefly to be of any value to a beginner in surgery. On the whole the book is an excellent one and should be well received.

1. *Modern Surgery: General and Operative.* By John Chalmers DaCosta, M. D., etc. Seventh edition, revised, enlarged and reset. Illustrated. $6.
2. *Infant Feeding.* By Clifford G. Grulee, M. D., etc. Second edition. Illustrated. $3.
3. *Psychanalysis: Its Theories and Practical Application.* By A. A. Brill, M. D., etc. Second edition, thoroughly revised. $3.00.

(*Philadelphia and London: W. B. Saunders Company, 1914.*)

These new editions are all valuable and can be warmly recommended to the profession. DaCosta's Surgery is only one of many on the market, but one of the best, and one that a real student of the art will find especially interesting, as the author frankly acknowledges his indebtedness to his predecessors and associates, quoting them and their words frequently, and so leading the reader back to original sources.

Grulee has not modified his book essentially, but gives the latest information to be had on questions of digestion that are being actively studied by many workers.

Brill has introduced new chapters and a glossary into his work, and in other ways also has made it more useful as a practical work.

Thomas Shortt. By ARNOLD CHAPLIN, M. D. Price 2s. (*London: Stanley Paul & Co., 1914.*)

This book contains a biography of Doctor Thomas Shortt, principal medical officer in St. Helena, and short biographies of some other medical men associated with the case of Napoleon from 1815 to 1821.

Written in the same fluent style as "The Illness and Death of Napoleon" by the same author, this work is most interesting and throws many side-lights on the trials and troubles of the medical advisors of Napoleon during his last illness.

Besides Thomas Shortt, short biographies are given of Doctors Geo. Henry Rutledge, Walter Henry, James Roche Verling, Archibald Arnott, Barry O'Meara, Francis Burton and Alexander Baxter.

Principles of Surgery. By W. A. BRYAN, A. M., M. D. Professor of Surgery and Clinical Surgery at Vanderbilt University, Nashville, Tennessee. Original Illustrations. (*Philadelphia: W. B. Saunders Company, 1913.*)

The volume is divided into 47 chapters in which the principles of surgery are thoroughly covered. The descriptions are short and clearly written. The book is well printed and the illustrations are good. The student and physician will find this up to date work a valuable help in the way of sound, practical surgical principles.

Health Through Diet. By KENNETH G. HAIG, L. R. C. P. (*Philadelphia: J. B. Lippincott Company, 1914.*)

Health, according to the writer, is to be obtained and secured by a uric-acid-free diet. We wish the problem were as simple as it seems to Dr. Haig, but that uric acid is such a powerful poison to the system, as is implied by him, is not generally conceded by other students of metabolism, and the author's work is founded only on personal experience. While such experience has value, yet it cannot take the place of experimental work on digestion, and Dr. Haig's statements lack convincing force. As a contribution to medicine the book has but little value.

Pathfinders of Physiology. By J. H. DEMPSTER, M. D. (*Published by the Detroit Journal Company, 1914.*)

This is a series of sketches, which may serve as a guide to students who are interested in studying the history of physiology. It is but a small volume of light but readable character. This volume cannot take the place of the classic work on this subject by Sir Michael Foster, which has been most inspiring to all students. It is to be hoped that Dr. Dempster's little book may lead the reader to more extended studies.

How to Diagnose Smallpox. By W. McC. WANKLYN, M. R. C. S., etc. $1.50. (*New York: Paul B. Hoeber, 1914.*)

There is much of value to the student in this small volume. The author presents his subject in an unfortunate style, but his experience has been large, and what he says is well worth reading; he does not lay much stress on the almost classical symptoms of pain in the back, the shotty feel of the papules, and the fact that all the papules show at any one moment but one stage of development, but lays more stress on the distribution and nature of the primary rash. There is no doubt that these latter points are very important. As a brief presentation of the subject the book offers many points of interest.

Map Scheme of the Sensory Distribution of the Fifth Nerve (Trigeminus) with its Ganglia and Connections. With Text. $8. By L. HEMINGTON PEGLER. (*New York: Paul B. Hoeber, 1914.*)

In colors, well designed and mounted on heavy cloth, this large folding map, 48 by 56 inches, would be serviceable if not bound into covers. It could then be used satisfactorily on the wall of a class room. As it is, it is unhandy and cannot be conveniently referred to.

Ambidexterity and Mental Culture. By H. MACNAUGHTON-JONES, M. D. 75 cents. (*New York: Rebman Company, 1914.*)

This book on a neurological topic by an obstetrician can lay little claim to scientific value, but is rather a rambling plea for the training of children in the equal use of the two hands, the plea being supported by the opinions of several authorities and extended by a quite irrelevant chapter on the Montessori method.

Diseases of the Skin. By GEORGE THOMAS JACKSON, M. D. 7th Edition. Price $3. (*Philadelphia: Lea & Febiger, 1914.*)

This new edition of Jackson's work appears with a marked change in binding, from a dark green to an aggressive red. This prepared us for a thorough revision of the contents, and we were not disappointed.

Much useless matter has been omitted, and new sections on fifteen diseases incorporated, in addition to revision of many old sections; which brings the book up to date.

Along special line of treatment, the author has been fortunate in securing the methods of men eminent in their line of work.

Quite valuable additions are two colored plates and sixteen very good half tones, but there remain a number of illustrations which serve no purpose, and should be replaced by others. The value of the book is greatly enhanced by the very thorough revision.

BOOKS RECEIVED.

A Mind Remedy. By John G. Rayerson, M. D. 1914. 16°. 82 pages. The Quinn A. Boden Company, Rahway, N. J.

American Association for Study and Prevention of Infant Mortality. Transactions of the Fourth Annual Meeting, Washington, D. C., November, 14-17, 1913. 1914. 8°. 447 pages. Franklin Printing Company, Baltimore.

Surgery. Its Principles and Practice. By Astley Paston Cooper Ashhurst, A. B., M. D., F. A. C. S. With 7 colored plates and 1032 illustrations in the text, mostly original. 1914. 8°. 1141 pages. Lea & Febiger, Philadelphia and New York.

A History of Laryngology and Rhinology. By Jonathan Wright, M. D. Second edition, revised and enlarged. 1914. 8°. 357 pages. Lea & Febiger, Philadelphia and New York.

Arteriosclerosis. A Consideration of the Prolongation of Life and Efficiency after Forty. By Louis Faugères Bishop, A. M., M. D. 1914. 8°. 383 pages. Henry Frowde, London; Hodder & Stoughton, London; Oxford University Press.

Guide to the Microscopic Examination of the Eye. By Professor R. Greeff. With the co-operation of Professor Stock and Professor Wintersteiner. Translated from the third German edition by Hugh Walker, M. A., M. B., C. M. [1913.] 4°. 86 pages. The Ophthalmoscope Press, London; Paul B. Hoeber, New York.

Asthma and its Radical Treatment. By James Adam, M. A., M. D., F. R. F. P. S. With four illustrations. 1913. 12°. 184 pages. Paul B. Hoeber, New York.

The Evolution of the Study of Anatomy and its Important Relation to the Development of Surgical Knowledge. By J. Ewing Mears, M. D., LL. D. 1914. 8°. 26 pages. Philadelphia.

Pathfinders of Physiology. By J. H. Dempster, A. B., M. D. 1914. 8°. 66 pages. The Detroit Journal Company.

How to Diagnose Smallpox. By W. McC. Wanklyn, B. A. Cantab., M. R. C. S., L. R. C. P., D. P. H. With illustrations. 1914. 8°. 104 pages. Paul B. Hoeber, New York.

The Midwife in England. Being a Study in England of the Working of the English Midwives Act of 1902. By Carolyn Conant Van Blarcom, R. N. With an introduction by J. Clifton Edgar, M. D. December, 1913. Prevention of Blindness, No. 13. 8°. 140 pages. New York City.

Map Scheme of the Sensory Distribution of the Fifth Nerve (Trigeminus) with its Ganglia and Connections. By L. Hemington Pegler. With text. 1913. Baillière, Tindall and Cox, London; Paul B. Hoeber, New York.

The Road to a Healthy Old Age. Essays Lay and Medical. By Thomas Bodley Scott. 1914. 16°. 104 pages. Paul B. Hoeber, New York.

Monographs on Biochemistry. Edited by R. H. A. Plimmer, D. Sc., and F. G. Hopkins, M. A., M. B., D. Sc., F. R. S. *The Simpler Natural Bases.* By George Barger, M. A., D. Sc. 1914. 8°. 215 pages. Longmans, Green & Co., London, New York, Bombay and Calcutta.

Health Through Diet. A Practical Guide to the Uric-Acid-Free Diet. Founded on Eighteen Years' Personal Experience. By Kenneth G. Haig, L. R. C. P., Lond.; M. R. C. S., Eng. With the Advice and Assistance of Alexander Haig, M. A., M. D., Oxon. 1914. 12°. 227 pages. J. B. Lippincott Company, Philadelphia; Methuen & Co., Ltd., London.

The Ready Reference Hand-Book of Diseases of the Skin. By George Thomas Jackson, M. D. With 115 illustrations and 6 plates. Seventh edition, thoroughly revised. 1914. 12°. 770 pages. Lea & Febiger, New York and Philadelphia.

Psychanalysis: Its Theories and Practical Application. By A. A. Brill, Ph. B., M. D. Second edition, thoroughly revised. 1914. 8°. 393 pages. W. B. Saunders Company, Philadelphia and London.

Modern Surgery, General and Operative. By John Chalmers DaCosta, M. D., LL. D. Seventh edition, revised, enlarged and reset with 1085 illustrations, some of them in colors. 1914. 8°. 1515 pages. W. B. Saunders Company, Philadelphia and London.

Infant Feeding. By Clifford G. Grulee, A. M., M. D. Illustrated, second edition, thoroughly revised. 1914. 8°. 313 pages. W. B. Saunders Company, Philadelphia and London.

Bedside Hæmatology: An Introduction to the Clinical Study of the So-called Blood Diseases and of Allied Disorders. By Gordon R. Ward, M. D. (Lond.). 1914. 8°. 394 pages. W. B. Saunders Company, Philadelphia and London.

Diseases of the Heart. By John Cowan, D. Sc., M. D., F. R. F. P. S. With Chapters on the Electro-Cardiograph. By W. T. Ritchie, M. D., F. R. C. P., and the Ocular Manifestations in Arterio-Sclerosis. By Arthur J. Ballantyne, M. D., F. R. F. P. S. 1914. 8°. 438 pages. Lea & Febiger, Philadelphia and New York.

Contribution to the Knowledge of Colloidal Nitrogen in Urine. Thesis. By Pieter Johannes Louis de Bloeme. Translation from the Dutch. 1914. 8°. 86 pages. J. H. de Bussy, Amsterdam.

Nucleic Acids, Their Chemical Properties and Physiological Conduct. By Walter Jones, Ph. D. 1914. 8°. 118 pages. Longmans, Green & Co., London, New York, Bombay and Calcutta.

Progressive Medicine. A Quarterly Digest of Advances, Discoveries and Improvements in the Medical and Surgical Sciences. Edited by Hobart Amory Hare, M. D., assisted by Leighton F. Appleman, M. D. Volume II. June, 1914. 8°. 460 pages. Lea & Febiger, Philadelphia and New York.

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